MDCT EVALUATION OF NON-TRAUMATIC ACUTE ABDOMEN

Submitted in partial fulfillment for

M.D. DEGREE EXAMINATION

BRANCH - VIII , RADIO DIAGNOSIS

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

COIMBATORE – 14

Dissertation submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600 032

TAMILNADU

APRIL 2017

CERTIFICATE

This dissertation titled **"MDCT EVALUATION OF NON-TRAUMATIC ACUTE ABDOMEN"** is submitted to The Tamilnadu Dr.M.G.R Medical University, Chennai, in partial fulfillment of regulations for the award of M.D. Degree in Radio Diagnosis in the examinations to be held during April 2017.

This dissertation is a record of fresh work done by the candidate **Dr. P. P. BALAMURUGAN,** during the course of the study (2014 - 2017).

This work was carried out by the candidate himself under my supervision.

GUIDE:

Dr.N.MURALI, M.D.RD,

Professor & HOD,

Department of Radio Diagnosis,

Coimbatore Medical College,

Coimbatore – 14

HEAD OF THE DEPARTMENT:

Dr.N.MURALI, M.D.RD,

Professor & HOD

Department of Radio Diagnosis,

Coimbatore Medical College,

Coimbatore – 14

DEAN:

Dr. A. EDWIN JOE, M.D, BL.,

Dean,

Coimbatore Medical College and Hospital,

Coimbatore – 14.

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : DR. BALAMURUGAN

Course : M.D. RADIODIAGNOSIS

Period of Study : 2014 - 2017

College : COIMBATORE MEDICAL COLLEGE

Dissertation Topic : MDCT EVALUATION OF NON-TRAUMATIC ACUTE ABDOMEN.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not~~ accepted and you are permitted / ~~Not~~ permitted to proceed with the above Study.

DEAN

Coimbatore Medical College & Hospital, Coimbatore

22.7.2015

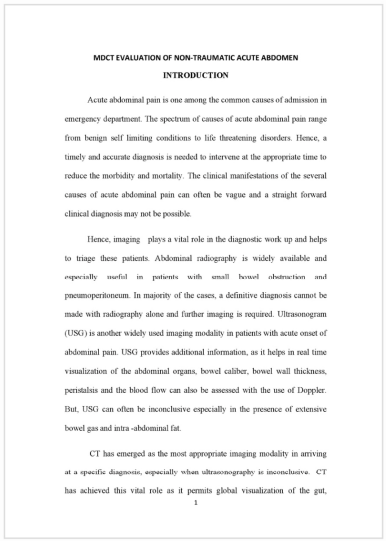
turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201418151 M.d.(radio-diagnosis) P..
Assignment title:	2015-2015 plagiarism
Submission title:	MDCT EVALUATION OF NON TRA...
File name:	KB_913_-_Copy.docx
File size:	2M
Page count:	85
Word count:	13,840
Character count:	78,266
Submission date:	23-Sep-2016 01:45AM
Submission ID:	704860033



MDCT EVALUATION OF NON-TRAUMATIC ACUTE ABDOMEN

INTRODUCTION

Acute abdominal pain is one among the common causes of admission in emergency department. The spectrum of causes of acute abdominal pain range from benign self limiting conditions to life threatening disorders. Hence, a timely and accurate diagnosis is needed to intervene at the appropriate time to reduce the morbidity and mortality. The clinical manifestations of the several causes of acute abdominal pain can often be vague and a straight forward clinical diagnosis may not be possible.

Hence, imaging plays a vital role in the diagnostic work up and helps to triage these patients. Abdominal radiography is widely available and especially useful in patients with small bowel obstruction and pneumoperitoneum. In majority of the cases, a definitive diagnosis cannot be made with radiography alone and further imaging is required. Ultrasonogram (USG) is another widely used imaging modality in patients with acute onset of abdominal pain. USG provides additional information, as it helps in real time visualization of the abdominal organs, bowel caliber, bowel wall thickness, peristalsis and the blood flow can also be assessed with the use of Doppler. But, USG can often be inconclusive especially in the presence of extensive bowel gas and intra -abdominal fat.

CT has emerged as the most appropriate imaging modality in arriving at a specific diagnosis, especially when ultrasonography is inconclusive. CT has achieved this vital role as it permits global visualization of the gut,

1

MDCT EVALUATION OF NON

BY 201418151 M.D.(RADIO-DIAGNOSIS) P.P.BALAMURUGAN

turnitin

15% SIMILAR

-- OUT OF 0

MDCT EVALUATION OF NON-TRAUMATIC ACUTE ABDOMEN

INTRODUCTION

Acute abdominal pain is one among the common causes of admission in emergency department. The spectrum of causes of acute abdominal pain range from benign self limiting conditions to life threatening disorders. Hence, a timely and accurate diagnosis is needed to intervene at the appropriate time to reduce the morbidity and mortality. The clinical manifestations of the several causes of acute abdominal pain can often be vague and a straight forward clinical diagnosis may not be possible.

Hence, imaging plays a vital role in the diagnostic work up and helps to triage these patients. Abdominal radiography is widely available and especially useful in patients with small bowel obstruction and pneumoperitoneum. In majority of the cases, a definitive diagnosis cannot be made with radiography alone and further imaging is required. Ultrasonogram (USG) is another widely used imaging modality in patients with acute onset of abdominal pain. USG provides additional information, as it helps in real time visualization of the abdominal organs, bowel caliber, bowel wall thickness, peristalsis and the blood flow can also be assessed with the use of Doppler. But, USG can often be inconclusive especially in the presence of extensive bowel gas and intra -abdominal fat.

Match Overview

1	radiographics.rsna.org Internet source	2%
2	Lassandro, Francesco... Publication	1%
3	geiselmed.dartmouth.... Internet source	1%
4	Cross-Sectional Imagi... Publication	1%
5	Submitted to Weber St... Student paper	1%
6	Radiology Illustrated U... Publication	1%
7	Levy, A. D., and C. M. ... Publication	<1%
8	"Abstracts from the 36t... Publication	<1%
9	Radiology Illustrated, 2... Publication	<1%

Text-Only Report

DECLARATION

I, **Dr. P.P. Balamurugan,** solemnly declare that the dissertation titled **"MDCT EVALUATION OF NON-TRAUMATIC ACUTE ABDOMEN"** was done by me at Coimbatore Medical College, during the period from July 2015 to August 2016 under the guidance and supervision of **Dr. N. Murali, M.D.RD,** Professor, Department of Radio Diagnosis, Coimbatore Medical College, Coimbatore. This dissertation is submitted to the Tamilnadu Dr.M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch -VIII) in Radio Diagnosis.

I have not submitted this dissertation on any previous occasion to any University for the award of any degree.

Place:

Date: **Dr. P.P.BALAMURUGAN**

ACKNOWLEDGEMENT

I express my sincere thanks to our respected Dean, **Dr. A. EDWIN JOE, MD., BL.,** Coimbatore Medical College and Hospital, Coimbatore for permitting me to conduct this study.

I thank **Dr. R. MANI, M.D,** Vice Principal, Coimbatore Medical College, Coimbatore for his encouragement and suggestions in completing this study.

I am greatly indebted to the Head of the Department of Radio Diagnosis, **Professor Dr. N. MURALI, MDRD.,** who has always guided me, by example and valuable words of advice. He has always given me his moral support and encouragement throughout the conduct of the study and also during my entire post graduate course. I owe my sincere thanks to him.

I thank **Professor Dr. N. SUNDARI, MDRD.,** Department of Radio Diagnosis for her valuable advice, support, guidance and suggestions.

I thank **Dr. C. SUBHASHREE, MDRD., DNB.,** Associate Professor, Department of Radio Diagnosis for her valuable suggestions.

I thank **Dr. R. KANNADHASAN, DMRD, M.D.,** Assistant Professor, Department of Radio Diagnosis for his support throughout this study.

I would like to thank my all other beloved teachers, Department of Radio Diagnosis for their valuable opinion and help to complete this study.

I would grossly fail in my duty, if I do not mention my gratitude to my **subjects** who have volunteered to undergo the investigations for the study.

My sincere thanks to all my **fellow postgraduates, Dr.NAINA SURESH, Dr.S.KANAGA DURGA, Dr.X.INFANT PUSHPA VENISHA, Dr.C.KALAIVANI and Dr.P.RAMACHANDRAN** for their involvement in helping me in this work.

I'm grateful to my **wife, Mrs. C. DEVI, my sons B.HARSHA VARDHAN and B.VISHNU VARDHAN and my parents and parents- in-law, brothers and sisters** who helped me making this study a reality.

My heartful thanks to my friends **Dr.E.AMUDHAN ARAVIND and Mr.J.JEBIN** for their help. Without their help and support this thesis could not be possible.

Above all I thank the **Lord Almighty** for His kindness.

CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF CHARTS

LIST OF ABBREVIATIONS USED

CT	–	Computed Tomography
NECT	–	Non Enhanced Computed Tomography
CECT	–	Contrast Enhanced Computed Tomography
MDCT	–	Multi Detector Computed Tomography
USG	–	Ultrasonogram
mIP	–	Minimum Intensity Projection
MIP	–	Maximum Intensity Projection
IV	–	Intra Venous
SBO	–	Small Bowel Obstruction
AMA	–	Aorto Mesentric Angle
SMA	–	Superior Mesentric Artery Syndrome
PI	–	Pneumatosis Intestinalis
GJ	–	Gastro Jejunal
APFC	–	Acute Peri Pancreatic Fluid Collection
ANC	–	Acute Necrotic Collection
PCS	–	Pelvi Calyceal System
GB	–	Gall Bladder
eGFR	-	Estimated Glomerullar Filtration Rate

INTRODUCTION

Acute abdominal pain is one among the common causes of admission in emergency department. The spectrum of causes of acute abdominal pain range from benign self limiting conditions to life threatening disorders. Hence, a timely and accurate diagnosis is needed to intervene at the appropriate time to reduce the morbidity and mortality. The clinical manifestations of the several causes of acute abdominal pain can often be vague and a straight forward clinical diagnosis may not be possible.

Hence, imaging plays a vital role in the diagnostic work up and helps to triage these patients. Abdominal radiography is widely available and especially useful in patients with small bowel obstruction and pneumoperitoneum. In majority of the cases, a definitive diagnosis cannot be made with radiography alone and further imaging is required. Ultrasonogram (USG) is another widely used imaging modality in patients with acute onset of abdominal pain. USG provides additional information, as it helps in real time visualization of the abdominal organs, bowel caliber, bowel wall thickness, peristalsis and the blood flow can also be assessed with the use of Doppler. But, USG can often be inconclusive especially in the presence of extensive bowel gas and intra -abdominal fat.

CT has emerged as the most appropriate imaging modality in arriving at a specific diagnosis, especially when ultrasonography is inconclusive. CT has achieved this vital role as it permits global visualization of the gut,

mesentry, omemtum, peritoneum, retroperitoneum, vasculature, solid organs, abdominal musculature and bones.

The purpose of the study is to evaluate the accuracy of CT in diagnosis of acute abdomen, to determine the impact of early CT diagnosis on clinical decision making regarding management, besides enumerating the spectrum of causes of non – traumatic acute abdomen.

OBJECTIVES OF THE STUDY:

1. To evaluate the accuracy of CT in the diagnosis of non traumatic acute abdomen in cases where USG and X-Ray findings are negative / non - specific or are unable to provide additional information relating to the diagnosis.
2. To evaluate the impact of CT in early diagnosis on the management of non-traumatic acute abdomen.
3. To enumerate the spectrum of causes of non-traumatic acute abdomen.

METHODOLOGY

TOSHIBA Multi-slice CT (4 slice) was used for all the cases. Serial axial section of abdomen and pelvis were taken from diaphragm to inferior border of symphysis pubis with a collimation of 5 – 7 mm and pitch of 1 to 1.5 depending on the length of coverage.

Multi-planar reconstruction was done at intervals of 3-7 mm. Axial and coronal/sagittal reformatted images were studied. When appropriate, maximum intensity projection, minimum intensity projection and volume rendering techniques were also analysed.

Initially plain CT abdomen and pelvis axial sections were taken, followed by contrast study. Iodinated I V contrast was routinely used except in patients suffering from medical renal disease and known anaphylaxis to medications. e GFR was calculated and contrast was administrated only when e GFR was normal. Oral and rectal contrast were used wherever necessary.

The I V Contrast used was IOHEXOL (Omnipaque) 350 mg iodine/ml at a dose of 1.75 ml /kg (Avg -90 to 100 ml) by using power injector through IV cannula (18 Gauge) at a rate of 2ml /sec.

STUDY DESIGN:

This was a prospective study of consecutive patients with acute abdomen in the study period from July 2015 to August 2016. The study was commenced after approval from the ethical committee. Formal consent for the study was obtained from all the patients.

INCLUSION CRITERIA:

H/O acute abdominal pain

H/O abdominal distension

Abdominal guarding and rigidity

Diagnosis made by ultrasonogram but CT requested by referring clinician for additional information.

EXCLUSION CRITERIA:

H/O trauma (Blunt injury and penetrating injury)

Pregnant mothers

Confirmed diagnosis made by ultrasonogram

REVIEW OF LITERATURE

The spectrum of causes of acute abdominal pain range from benign self-limiting conditions to life threatening disorders. Abdominal radiography and USG form the initial imaging modalities. CT has emerged as the most appropriate imaging modality in arriving at a specific diagnosis, especially when ultrasonography is inconclusive. The spectrum of causes of non traumatic acute abdomen and their imaging findings in CT are described below.

I. DISEASES OF BOWEL, PERITONEUM & OMENTUM:

1. ACUTE APPENDICITIS

Appendicitis constitutes the most frequent cause of acute abdominal pain requiring surgical intervention. Appendicitis occurs mostly occurs due to luminal obstruction by fecoliths, lymphoid hyperplasia, tumors, parasites, foreign body, stricture. Among the above mentioned causes, fecoliths constitute the most common cause. Apart from clinical assessment, radiological findings play a major role in diagnosing appendicitis and excluding other causes that mimic appendicitis thereby reducing negative appendicectomy rate. The sensitivity and specificity of CT in diagnosis of appendicitis are 94% and 95% respectively[1].

Appendix measuring more than 6mm diameter has a high positive predictive value in diagnosing appendicitis[2]. But the diameter is alone not sufficient to diagnose appendicitis. The presence of associated wall thickening, appendiculolith, periappendiceal fat stranding, enhancement after

administration of IV contrast and the presence of fluid or abscess on CT points towards diagnose of appendicitis.

In early and mild cases, the appendix appears fluid filled, minimally distended and measuring about 5 – 6 mm diameter, peri - appendicial fat may be normal at this stage. As disease progresses, the appendix progressively distends and the wall thickens and shows enhancement on administration of intravenous contrast. Mural enhancement pattern can be homogeneous or may have a target sign appearance due to serosal, mucosal enhancement and sub mucosal edema. Periappendicial fat stranding is visualized. Secondary reactive thickening of wall of terminal ileum or caecum can be seen.

In perforated appendix, pockets of extra luminal air can be seen with marked thickening of ileo-caecal wall, lymphadenopathy, peri-caecal phlegmon or abscess with localized peritonitis.

Following oral contrast study, the **caecal arrow head sign and caecal bar sign** can be appreciated. The CT arrow head sign refers to triangular configuration of oral contrast funnelling in to focally thickened caecum pointing towards appendiceal orifice. The caecal bar sign is due to the linear inflammatory soft tissue that separates contrast filled caecum from appendix.

Recurrent appendicitis occurs when appendicectomy is done with simple ligation of stump without stump invagination. The residual stump behaves as a small appendix or diverticulum, which gets inflamed.

Distal appendicitis shows a normal caecal apex and proximal appendix with gradual or abrupt enlargement of distal appendix with surrounding periappendicial inflammatory changes.

2. SMALL BOWEL OBSTRUCTION

In patients suspected or known to have small bowel obstruction, imaging plays a critical role in diagnosing the level of obstruction, determining the cause and severity of obstruction.

Simple obstruction indicates that the lumen of bowel is partly or completely occluded, perhaps the blood supply is preserved. When the blood supply to the bowel is reduced, it is termed as strangulation. Small bowel obstruction can also be classified as open loop or closed loop obstruction. In open loop obstruction, the bowel is obstructed distally but the bowel contents can be decompressed proximally. In closed loop obstruction the segment of bowel loop is obstructed both proximally and distally resulting in increased intra luminal pressure and increased risk of strangulation. CT provides a non invasive method of assessment of bowel wall, bowel lumen, adjacent mesentry and vasculature. It can also identify superimposed infection, ischemia and perforation.

Diagnosis of SBO in CT is indicated by the identification of transition zone, dilated air-fluid filled loops fluid filled loops proximal to obstruction, collapsed loops distal to the obstruction. The diameter of small bowel exceeds 2.5 cm and the dilatation should be continuous[3]. CT also helps to determine the

cause at the level of transition zone. Bowel wall can be minimally thickened or normal in thickness at level of obstruction. Mesenteric vascular engorgement, haziness and ascites are minimal in simple SBO.

Small bowel faeces sign is frequently seen in high grade obstruction in which particulate like material can be identified in dilated small bowel and is best appreciated at the point of transition[4].

CT findings in closed loop obstruction include the presence of focally dilated bowel loop, with a C-shaped or U-shaped or radial configuration depending upon the plane of reconstruction. The entering and exiting loop lay side by side at the point of entry and appeared narrowed and tapered at the site of entry. When the closed loop is twisted, the twisting of mesenteric vessels give the appearances of **Whirl's sign**[5] .The **serrated beak sign** refers to the presence of mesenteric vascular engorgement and bowel wall thickening at obstructed site.

Strangulation, mostly occurs as a complication of volvulus, torsion and closed loop obstruction. The presence of portal venous gas, mesenteric venous gas pneumatosis intestinalis are one of the most specific signs to diagnose strangulation[6]. Absent or poor contrast enhancement of bowel wall has specificity of 100% in diagnosing strangulation.

Adhesions and bands are most common cause of obstruction and are usually not visible on CT as they are fibrous and thin. But sometimes, **the extra luminal band sign**, which is a linear compression on the bowel in area

of transition is seen. Internal and external abdominal hernias constitute the next major cause of extrinsic SBO. CT is very helpful to demonstrate the site and content of hernial sac. SBO by internal hernia can be suspected when there is abnormal location and crowding of bowel loops and when the mesenteric vessels show an unsual course.

Bezoars appear as round, ovoid or sausage shaped material with mottled gas seen at obstructed site. These intra luminal filling defects can often be multiple. Bowel wall thickening is seen on CT at the site of obstruction or in the bowel loop proximal to the obstruction.

Gall stone ileus occurs when a larger gall stone 2.5 cm or more in diameter enters the small bowel via a choledocho-enteric or cholecysto-enteric fistula and obstructs the lumen, which is most commonly seen in the distal ileum. The gall stone in distal bowel with proximal dilatation of bowel loops and pneumobilia is known as **rigler's triad.**

Neoplastic, inflammatory lesions that are extrinsic to bowel can cause SBO as they compress and distort the bowel for eg: carcinomatous deposits orginating from ovarian, gastric, colonic or liver malignancy causing desmoplastic reaction, causing mass effect, angulation of bowel and luminal narrowing.

Enteric duplication cysts located at mesenteric border of distal ileum compresses it and leads to obstruction. It is seen as a soft tissue or fluid attenuation lesion at mesenteric border of ileum.

Intrinsic tumors and inflammatory lesions can affect the bowel wall and causes luminal narrowing. Primary tumors of intestine like adenocarcinoma cause annular narrowing with abrupt cut off. Inflammatory conditions such as crohn's disease and TB can cause strictures resulting in luminal narrowing. Terminal ileum is most commonly affected in crohn's whereas cecum and ascending colon are more commonly affected in TB. The ileo-caecal valve is often patulous in TB and is stenotic in crohn's disease.

3.SMA SYNDROME

SMA syndrome is a rare acquired vascular compression disorder. It's prevalence is around 0.1%–0.3%. The SMA arises from the aorta at the L1-2 level and it courses anteriorly and inferiorly, forming an angle with the aorta known as the **aorto-mesentric angle (AMA)**. The normal AMA measures 28-35 degrees[8]. The third portion of the duodenum crosses between the aorta and the proximal SMA at approximately the L3 level. The SMA is surrounded by retroperitoneal fat, which provides a "cushion" for the duodenum. The factors that predispose to the occurrence of SMA syndrome include, thin body build, exaggerated lumbar lordosis, visceroptosis and abdominal wall laxity, abnormally high and fixed position of the ligament of Treitz with an upward displacement of the duodenum, unusually low origin of the superior mesenteric artery

SMA syndrome commonly affects females in 10-40 years of age. They present with postprandial epigastric pain and fullness, nausea, vomiting, weight

loss and anorexia. These symptoms occur due to compression of the SMA on the third part of duodenum.

CECT is the diagnostic test of choice for diagnosis of SMA syndrome. It allows simultaneous evaluation of the mesoaortic vascular anatomy, transverse duodenal compression and proximal dilatation. It has the added advantage of showing the exact anatomic position of the duodenum in the vascular angle and excluding other causes of obstruction. A duodenal diameter of more than 3 cm is the indication of presence of duodenal dilatation. The **aorto-mesentric distance** is reduced measuring 2-8mm and aorto-mesentric angle is reduced measuring 6-22 degrees. The diagnosis of SMA syndrome should be made only when the there are clinical signs of duodenal obstruction along with the above mentioned CT features.

4.PERITONITIS

Peritonitis is an inflammatory condition of peritoneum, etiology of which could be infective or non infective. It can be grouped as primary and secondary peritonitis. Primary peritonitis is diffuse infection of peritoneal cavity without loss of integrity of GIT. Secondary peritonitis occurs following hollow viscus perforation, peritoneal dialysis. Non infective causes include eosinophilic peritonitis or encapsulating peritoneal sclerosis, biliary peritonitis. Appendicitis, crohn's disease, diverticulitis can result in localized peritonitis.

On MDCT the normal peritoneum appears as a fine thin structure and its hardly visible. Pathological conditions of peritoneum produce thickening which can have a smooth regular pattern, irregular pattern and nodular pattern.

Smooth regular pattern:

Localized peritonitis secondary to appendicitis, diverticulitis, crohn's disease is characterized by fluid collection around the inflammed organ with smooth peritoneal thickening that shows contrast enhancement.

Perforation of GI tract is associated with presence of extra luminal gas and free fluid in peritoneal cavity. Peritoneal thickening, segmental bowel wall thickening, peri-visceral fat stranding, abnormal bowel wall enhancement are other features that can be observed.

Spontaneous bacterial peritonitis is a type of primary peritonitis occurs due to infection by organisms like E.coli, streptococcus which is commonly seen in patients with cirrhosis. On CT, it appears as smooth thickening of peritoneum involving the whole abdominal cavity associated with presence of gross ascites with the mesenteric folds are often relatively spared.

Rupture of biliary duct, post operative biliary leak, or pathologic GB can result in biliary peritonitis. On CT, often a loculated fluid collection is seen at hepatic hilum and sub-hepatic space. Diffuse biliary peritonitis is associated with smooth peritoneal thickening.

Wet type of TB peritonitis causes smooth thickening of peritoneum which is associated with smudged appearance of omentum, thickened strands,

crowded vessels of mesentery[9]. Associated lymphadenopathy, splenomegaly with calcification and ileo-caecal wall thickening can be seen.

Irregular pattern:

Dry type TB peritonitis is associated with fibrous peritoneal reaction and dense adhesions presents with irregular peritoneal thickening, smudged appearance of omentum with necrotic lymph node, ileo-caecal wall thickening.

Encapsulating peritoneal sclerosis can be idiopathic or occurs in patients on ambulatory peritoneal dialysis. On CT, there is irregular peritoneal thickening and the small bowel loops lie towards the centre of abdomen. Peritoneal, serosal bowel wall, liver, splenic calcifications can be seen. Loculated fluid collections or gross ascites can be seen.

Nodular pattern:

Peritoneal carcinomatosis due to primary abdominal or extra abdominal tumor[10] shows diffuse peritoneal involvement which can encase the small bowel and involve the bowel resulting in perforation. Fibrotic fixed type of TB peritonitis shows omental mass formation and matted small bowel loops and nodular peritoneal thickening.

5.DIVERTICULITIS

Obstruction of the neck of diverticulum results in inspissation of fecal material resulting in distension and inflammation that causes diverticulitis and

extension of inflammation to the pericolic fat, further the wall of diverticulum can be eroded leading to peritonitis and fistula.

CT has sensitivity of 97% and sensitivity of 73% in diagnosing diverticulitis[11]. Diverticulae appear as small air filled outpouching with fecal material projecting through colonic wall and it is associated with thickening of colonic wall of more than 4 mm, when the colonic diameter is less than 1 cm. The presence of peri-colic fat inflammation is the hall mark of acute diverticulitis which is seen as fine linear strands, tiny bubbles of extra luminal air and engorgement of vasa-recta, tiny fluid collections can be seen. In severe cases heterogeneous soft tissue densities representing phlegmon can be present

Staging of diverticulitis on CT

Stage 0 – Most common form with inflammation being contained within serosa. It appears as mural thickening with inflammatory changes seen in surrounding fat.

Stage Ia - Defines peri-colic inflammation or phlegmon.

Stage Ib - Diverticulitis associated with peri-colic abscess. Abscess is managed conservatively when less than 3 cm and drained when more than 4cm.

Stage II - Diverticulitis associated with peri-colic abscess which has breached sigmoid mesocolon and is walled off by the omentum, small bowel. The abscess are usually larger 4 to 15 cm diameter and amendable to percutaneous drainage

Stage III - Diverticulitis with pelvic abscess that has spread beyond the pelvis to involve other portions of peritoneal cavity. CT demonstrated diverticulitis changes associated with localized or generalized fluid collection.

Stage IV- Diverticulitis with fecal spread into the peritoneal cavity. The findings are same as that of stage III but associated with features of acute peritonitis and sepsis.

6.EPIPLOIC APPENDAGITIS

Appendices epiploicae are sacs of visceral peritoneum filled with fat and contain vascular structures and they are located along the serosal surface of colon. These can undergo inflammatory changes, venous thrombosis and infarction and torsion, which is termed as primary epiploic appendagitis. Secondary epiploic appendagitis is caused by inflammation of adjacent organs.

CT is very useful to make diagnosis of this condition. These are seen as pericolonic oval shaped lesions with fat attenuation with periappendageal fat stranding with surrounding pareital peritoneal thickening[12]. There can be a central high attenuation dot within it, that represents thrombosed vessel. There can be mass effect on adjacent colon with adjacent focal wall thickening.

7.OMENTAL INFARCTION

Torsion of omental vessels, kinking or thrombosis of veins in omentum can result in vascular compromise resulting infarction of omentum. Primary infarction result from vascular compromise due to reduced blood supply to the right side of omentum due to kinking of veins[13]. Secondary omental infarction

is often due to surgery, trauma, hyper coagulable state, congestive cardiac failure or vasculitis[14].

It occurs most commonly in right iliac fossa and right hypochondrial region which could mimic acute appendicitis or cholecystitis. On CT, it appears as soft tissue or as heterogeneous large mass that contains variable amount of fat most commonly seen in right iliac fossa or hypochondrial region often 5 cm or larger in size[13].

Omental torsion occurs when a portion of omentum twists on itself and on CT twisting of vessels is seen. This condition is self limited and treated conservatively.

8.PNEUMATOSIS INTESTINALIS (PI)

Pneumatosis intestinalis refers to the presence of gas in the bowel wall. It is seen in association with various conditions ranging from benign to life threatening. Benign causes include pneumatosis cystoides intestinalis, pulmonary causes like asthma, cystic fibrosis, emphysema, other causes are sclerodema, SLE, enteritis, intestinal pseudo obstruction steroid therapy adynamic ileus, ulcerative colitis, crohn's disease. Life threatening causes include mesenteric vascular disease, strangulation, toxic mega colon, trauma and corrosive agent ingestion[15].

CT has superior diagnostic accuracy. PI appears as linear or bubbly patterns of gas in the bowel wall. The sub mucosal and sub serosal layers are most commonly involved.

The pattern of intramural gas alone do not allow differentiation between benign and life threatening causes. But the presence of associated findings such as bowel wall thickening, absent mucosal enhancement, dilated bowel, arterial or venous occlusion, portal venous gas increases the possibility of underlying life threatening causes[16].

9. INTUSSUSCEPTION

When a loop of intestine with its mesentry invaginates into lumen of bowel distal to it, the condition is known as intussusception. The advancing inner segment is termed as intussusceptum and the outer segment is known as intussuscepiens. Almost half of adult cases of intussusception are idiopathic the remaining half are due to benign polypoidal neoplasms, meckel's diverticulum, lymphoid hyperplasia, adhesions, coeliac disease, small bowel malignancy, peutz-jegher's syndrome. Colocolic intussusception are more likely due to malignant etiology[17]. In HIV positive patients, ileocolic intussusception are more likely due to lymphoma, atypical mycobacterial infection, kaposi sarcoma, mesenteric adenopathy. Appearance of intussusception on CT depends upon the plane of scanning axis. Intussusception appears as concentric rings of soft tissue and it has eccentric fat located within the lumen of outer ring. It can appear as sausage shaped or reniform mass with alternating layers of low and high attenuation. When vascular compromise develops due to compression or torsion of vessels between the layers of bowel, intestinal infarction develop. On CT, it appears as loss of layered bowel patterns with vascular engorgement and twisting of

mesenteric vessels **(whirl's sign)** extra luminal fluid collection or bowel perforation.

10.JEJUNOGASTRIC INTUSSUSCEPTION

There are 3 types of jejuno-gastric intussusception. The incidence of jejuno-gastric intussusception following jejuno-gastric anastomosis is 0.1%[18]. Retrograde peristalsis is proposed the main factor contributing to intussusception. There are three types of GJ intussusception[19]

Type I - Afferent loop alone may intussuscept into stomach also known as descending intussusception.

Type II - The efferent loop can undergo retrograde intussusception, also called ascending intussusception is the more common type . It constitutes for about 74% of the total cases of GJ intussusception.

Type III – both afferent and efferent loops together can intusscept into stomach known as combined type. The diagnosis of this condition is important as it carries a mortality of 80%. Retrograde intussusception of jejunal segment into the gastric remnant could be acute, chronic or transient. On CT the gastric remnant can be dilated and the proximal jejunum, adjacent mesenteric vessels area fat can be seen to enter the lumen of the stomach .

11.ACUTE MESENTERIC ISCHEMIA

It is characterized by sudden onset of severe abdominal pain disproportionate to clinical examination. This condition could be fatal and has

a very high mortality[20]. CT plays a major role in its diagnosis. The ischemic bowel appears thickened with lack of enhancement and pneumatosis can be seen.

This condition can be broadly grouped into four categories

1. Embolism - Most common cause of acute mesenteric ischemia being dislodgment of embolism from cardiac chambers. It mostly lodges at branch points of mid and distal SMA distal to middle colic artery. On angiogram occlusive thrombus is seen as termination of vessel known as **cut off sign.**
2. Arterial thrombosis - It occurs due to severe atherosclerotic changes. But since the condition slowly progressive there is adequate time for development of collaterals. Ischaemia occurs when there is complete occlusion of lumen of the distal arteries supplying the bowel. The occlusion usually involves the ostia and proximal 2 cm of SMA.
3. Non occlusive cause – This condition occurs due to cardiac dysfunction, hypervolemia, hypotension, renal and hepatic disease[21] and the involved segment of bowel shows lack of contrast enhancement. The mesenteric vessels appear diffusely thin in caliber. The watershed zone like splenic flexure of colon is commonly involved.
4. Venous thrombosis – Post operative status, portal hypertension, inflammatory conditions in abdomen and pelvis, oral contraceptive use, hyper-coagulable states are the most important causes. SMV is most

commonly affected, the affected segment shows bowel wall edema, hemorrhage, and sloughing of mucosa.

12.HERNIA

Abdominal hernias are defined as protrusion of part of its content from abdominal cavity through a normal or abnormal aperture or from the wall weakness[22] .

Hernias can be broadly grouped into internal and external hernias.

External hernias

Indirect inguinal hernias protrude through patent internal inguinal ring lateral to inferior epigastric vessels. Direct hernias protrude through the weak posterior wall of inguinal canal medial to inferior epigastric vessels[22]. Femoral hernias pass medial to femoral vein. Femoral hernia have the highest risk of going for obstruction as compared to other inguinal hernias.

Ventral group of hernias can be classified as anterior and lateral abdominal wall hernias. Anterior group includes umbilical, paraumbilical, epigastric and hypogastric hernias[23].

Umbilical hernias in adults are often due to acquired causes. Most common acquired causes of umbilical hernia are obesity and multi parity. These are usually asymptomatic, but they can be complicated by obstruction or strangulation.

Epigastric hernias are ventral hernias occurring in midline above umbilicus due to defect in linea alba. Hypogastric hernias are those which occur below umbilicus. They usually contain properitoneal fat and vessels and rarely solid viscera. Midline hernias are often complicated by obstruction, strangulation and incarceration.

The incisional hernias usually occur at sites of incision made for previous surgeries. They occur in midline or para median region. They often contain omentum and small bowel. One type of incisional hernia parastomal hernia which occurs around the stoma site, often contains fat, greater omentum which can also become incarcerated or strangulated.

Spigelian hernia is a type of lateral hernia which occurs due to weakness of posterior layer of transversalis fascia along the semilunar line between rectus sheath and oblique muscles. It usually contains omentum and short segments of small or large bowel loops. It frequently goes for incarceration .

Posterior hernias may occur through defects in lumbar muscle or in superior lumbar triangle of grynfeltt - lesshalf triangle or the inferior petit triangle. It may contain bowel loops or fat viscera. It can also go for obstruction and strangulation.

Less common hernias

Other less common hernias are sciatic, obturator and perineal hernias. Obturator hernia seems to occur between the obturator externus and pectineal

muscles or between the layer of obturator membrane it often contains bowel loops, bladder, uterus and adnexal tissue.

Sciatic hernias occur through sciatic foramen either above or below piriformis muscles and under inferior border of gluteus maximus. This hernia contains the small bowel or distal ureter. Perineal herinias occur in older women due to weakness of pelvic floor and they are seen near the anus, labia majora or gluteal region.

Diaphragmatic hernias

They can occur either through the hiatus (sliding or paraesophageal) or through lateral openings (anterior morgagni and posterior bochdelak hernias). Sliding hernias are the most common forms and constitute about 99% of all diaphragmatic hernias. These are caused by weak phrenico-esophageal membrane. Gastro-oesophageal junction is well above the diaphragm which predisposes it for reflux. Sliding hernias are complicated by incarceration and gastric volvulus[24]. Para esophageal hernias contain portions of stomach herniating into the chest wall while OG junction remains below diaphragm.

Internal hernias

Internal hernias involve protrusion of viscera within abdominal cavity through peritoneum / mesentery. They may present with symptoms of acute intestinal obstruction. These hernia may spontaneously resolve or incarcerate depending upon the size of formina and size of bowel loops. Internal hernias are of the following types[22].

1. Para duodenal hernia - 50 - 55 % of internal hernias.
2. Through foramen of winslow – 6 - 10%
3. Trans mesenteric hernias- 8 - 10%
4. Para caecal hernias – 10 - 15%
5. Intersigmoid hernias – 4 - 8%
6. Paravesical hernias - < 4%

Though hernias are not acute emergencies, these patients can present to the emergency department when they develop complications like incarceration, bowel obstruction, volvulus and strangulation. CT scan can help differentiating the palpable abdominal wall masses and hernia. It also helps find the hernia contents.

Even subtle signs of complications are evident on a CT. CT findings may include extravasated oral contrast material, bowel wall thickening and enhancement and bowel wall discontinuity.

13.VOLVULUS OF GIT

Volvulus of gastro intestinal tract is a twist of any segment of intestine containing mesenteric attachment. The patients present with abdominal pain, distension and vomiting. CT plays a major role in identification of volvulus and its complications.

Gastric volvulus

This type of volvulus is uncommon. Gastric volvulus is identified clinically by **borchardt triad** which consists of sudden epigastric pain,

intractable vomiting and inability to pass naso gastric tube into stomach[26]. Gastric volvulus can be organoaxial which is rotation along the long axis of stomach or mesentero-axial which in rotation along the short axis. Mesentero-axial is less common than organo-axial type. In organoaxial volvulus, greater curvature is displaced superiorly and lesser curvature displaced caudally. The antrum comes to lie in anterosuperior position where as the fundus is seen postero inferior. If the twist is more than 180 degree, gastric outlet obstruction occurs and stomach is distended with fluid. If positive oral contrast is used, the contrast is retained in the stomach.

Midgut volvulus

Midgut volvulus presents with acute onset of abdominal pain, nausea, vomiting. At CT, swirling of vessels at the mesenteric root is seen at the site of volvulus. There is altered relationship of superior mesenteric artery and vein. The abnormal position of ligament of trietz and the ectopic location of small bowel loops are identified. In severe cases, CT shows findings indicating obstruction and ischemia.

Colonic volvulus can occur at the level of caecum, transverse colon or sigmoid colon.

Caecal volvulus

Caecal volvulus accounts 25 – 40 % of all cases of colonic volvulus. This occurs due to rotation of right colon on its long axis and is usually associated with greater mobility of ascending colon. At CT, the abnormally

positioned dilated caecum is seen in upper and mid left side of abdomen with positive **whirl sign**.

Transverse colon volvulus

Transverse colon volvulus is the rarest, occurring in <5 – 10% of cases. It occurs due to abnormal fixation of long transverse colon. On CT or administration of contrast enema, there is beak like tapering of colon at level of twist.

Sigmoid volvulus

Sigmoid volvulus is the most common and constitutes about 60 – 75% of colonic volvulus. It occurs is the setting of high fibre diet, prolonged hospitalization or chaga's disease. CT shows the abnormal position of sigmoid volvulus above transverse colon which is called **northern exposure sign**. Swirling of mesentery at the site of twist is seen. Coronal reformation of CT is uses to visualize the U shaped closed loop appearance of dilated colon with **three line or white stripe sign** of opposed walls of dilated bowel loop[27]. If rectal contrast is given, beak shaped area is seen at the level of distal aspect of twist in the sigmoid beyond which the contrast cannot pass.

14.CROHN'S DISEASE

Crohn's disease is a chronic transmural inflammatory disorder that can involve the GIT from the mouth to anus. This disease commonly presents around the age of 20 years. The common sites being affected are ileocaecal area, distal small bowel and proximal ascending colon[29]. CT plays a major role

in diagnosis and in management of crohn's disease. The extent of involvement the bowel, the distribution of bowel involvement and extramural and mesenteric manifestations can be picked up in CT.

Increased attenuation in a non contracted bowel segment on contrast enhanced CT is on among the most consistent finding in crohn's disease. It may be bilaminar or trilaminar patterns. In bilaminar pattern, only the mucosa enhances. In trilaminar pattern, the mucosa and serosa enhance, but the intervening wall is hypo attenuating. The bowel wall enhancement can be transmural, asymmetric or patchy bowel wall thickening could be symmetric or asymmetric. It could be classified as mild (3-4 mm), moderate (5-10 mm) or severe (>10 mm). The proliferation of fibrous tissue or smooth muscle can result in luminal narrowing and cause proximal dilatation of small bowel loops. When the luminal narrowing is associated with upstream dilatation of small bowel of more than 3 cm, it is confirmed as stricture. Ulceration can be seen as wall defect that does not extend beyond the wall and if there is extension beyond the wall it is termed as sinus or fistulous tract and its is associated with angulation and separation of bowel loops. Fistula could be a simple fistula which extends from bowel loop to another or adjacent organ or it could be complex fistula associated with multiple tracts that involve several loops of bowel or adjacent organs. It can be seen in association with inflammatory mass or inter loop abscess.

Crohn's disease is also associated with mesenteric findings which include distension of mesenteric vessels that extend towards small bowel,

which is termed as **comb sign**[30,31,32]. Fibrofatty proliferation is associated with increased fat along mesenteric border of the small bowel leading to bowel loop separation.

Inflammatory mass presents as an ill defined, mass like soft tissue attenuation in the mesenteric fat and it is almost always associated with penetrating disease. Inflammatory abscess is a well formed thick walled fluid attenuation collection in mesenteric fat. CT can indentify, if the abscess is amendable to drainage by determining if the abscess can be entered without traversing bowel, vessels or bony structure.

Mesenteric lymph nodal enlargement in defined as nodes with short axis diameter of more than 1.5 cm. When the short axis diameter of the node is less than 2 cm it is more likely inflammatory and when the short axis diameter is more than 2 cm, of tumor should be reached[33].

15.ULCERATIVE COLITIS

Ulcerative colitis is a chronic mucosal inflammatory reaction which remains limited to mucosa and submucosa, which can involve the entire colon and terminal ileum. The complications of ulcerative colitis presents as acute abdomen which include perforation with perforative peritonitis, abscess or toxic megacolon. Toxic megacolon should be taken into consideration when colonic lumen is dilated considerably for a diameter of more than 8 cm. The bowel wall in toxic mega colon appears thinner as opposed to bowel wall thickening seen in chronic cases.

16.MECKEL'S DIVERTICULUM

Meckel's diverticulum is the most common congenital anomaly of GIT, the incidence being 2-3%. This occurs due to improper closure and absorption of omphalomesenteric duct. The most common complications are haemorrhage from peptic ulceration, small intestinal obstruction and diverticulitis[35].

The most frequent complication of meckel's diverticulum is haemorrhage[36], which occurs due to small amount of heterotopic gastric mucosa. TC -99 pertechnate is the most sensitive technique to identify haemorrhage. Angiography is used to localise the site of haemorrhage, which is seen as vascular blush. Angiography is useful for preoperative embolisation.

To identify meckel's diverticulum as the cause of intestinal obstruction is difficult with CT, but few signs that raise suspicion are described. It can be seen as blind ending tubular sausage shaped structure which communicates with small intestine lumen. Within the diverticulum, fluid, air, enteroliths, fecal like material can be seen associated with surrounding fat stranding or fluid in mesenteric fat.

Meckel's diverticulum may invaginate or invert into small intestinal lumen which could serve as the site of intestinal obstruction or lead point of intussception[37]. On CT, there will be a central core of fat attenuation surrounded by soft tissue attenuation.

Meckel's diverticulitis is due to obstruction or narrowing of its mouth by an enterolith, fecoliths, neoplasm or inflammation. On CT, diagnosis

lies on the identification of blind ending tubular structure in right lower quadrant or in periumbilical region with mural thickening and contrast enhancement[39]. Surrounding fat stranding is also noted. Sometimes, enteroliths are also visualised. The diverticulum extends upto umbilicus when it is attached by a fibrosis cord. The clues to diagnosis are pouch like structure attached to small intestine with secondary small intestinal obstruction and visualisation of normal appendix.

17.INTRA ABDOMINAL ABSCESS

CT plays a major role in detection of abscess, localizing it to specific anatomical location and aids in image guided drainage[40]. CT has a sensitivity and specificity of 97% and 95% respectively in diagnosing intra abdominal abscess, as compared to USG which has sensitivity and specificity of 82% and 94.5% respectively.

On CT, abscess can have irregular or well defined margins and may contain septations. The abscess are of fluid density but the attenuation value can be higher due to presence of blood, debris, proteinaceous material. Gas can be seen in the abscess, which could be seen in the form of microbubbles or as air fluid levels. The presence of gas within the abscess could indicate gas forming organisms such as E.coli, klebsiella, clostridia to be the cause. The presence of air fluid levels should raise the suspicious of fistulous connection with gastrointestinal tract.

The localization of abscess to specific anatomic location helps to indentify the cause that had lead to the formation of abscess.

Right **subdiaphragmatic space** communicates with right sub hepatic space and paracolic gutter. Inflammatory processes in these locations for eg: appendicitis, perforated viscus or perforated GB can result in formation of abscess. In left sub phrenic abscess, the sites of orgin are from the stomach, colon, or rarely esophagus. It can occur due to perforating ulcer, postoperative leaks or a neoplasm that has resulted in perforation.

Right **subhepatic space** located between undersurface of right lobe of liver and anterior surface of right kidney. Functionally, it can be divided into anterior and posterior parts depending on causative factor. Gall bladder perforation is most common cause for abscess formation in anterior compartment. The posterior compartment is the most dependent portion in supine position, hence fluid can collect from paracolic gutters or subphrenic space.

Omental bursa can be involved by inflammation extending from the pancreas or penetrating ulcer in posterior wall of stomach. Inflammation can reach omental bursa through foramen of winslow.

Pericolic abscess occurs as a result of inflammatory process following perforated colon , appendix or ruptured GB.

The **pelvis** forms the most dependent portion of abdomen, appendicitis and diverticulitis are the most common causes.

Posterior pararenal space lies between transversalis fascia and posterior renal fascia, inflammatory process extending into this space include ruptured aortic aneurysm, pancreatitis, osteomyelitis from rib and vertebrae.

Anterior pararenal space likes between anterior renal fascia and posterior parietal peritoneum, inflammatory process from pancreas, duodenum, colon, appendix is seen to extend into this space.

II.DISEASES OF RETROPERITONEUM:

1.ILIO-PSOAS ABSCESS

The ilio psoas compartment is composed of a group of extra peritoneal muscles that extend from posterior mediastinum to the hip joint. Pyogenic psoas abscess are usually secondary to spread from adjacent inflammatory disease. Most of these are due to direct spread from spinal or epidural infections, bowel infections such as crohn's disease, diverticulitis , appendicitis, perforated colonic carcinoma and perinephric abscesses. Primary abscess rarely occur and are usually idiopathic. Most common associated organisms are Staphylococcus aureus and mixed gram negative organisms.

On CT scan, they are seen as enlargement of ilio-psoas muscle[40] by a lesion of low attenuation. On IV contrast study, rim enhancement of the lesion is seen. Other findings include obliteration of surrounding tissue planes by inflammation and at times gas bubbles and bone destruction .

2.ILIO-PSOAS HAEMORRHAGE

Ilio-psoas haemorrhage may be secondary to trauma or it can occur spontaneously as in bleeding diathesis and anticoagulant therapy. On CT, ilio-psoas haemorrhage can appear hyperattenuating on NECT and can have fluid-fluid levels.

III.DISEASES OF LIVER:

1.LIVER ABSCESS

Pyogenic abscess – Bacteria reach the liver, through haematogeneous route or by direct infection that spreads from adjacent structures[41] and can result in abscess formation. E.coli and klebsiella are the most common species.

NECT – there is hypodensity due to presence of pus which is seen predominatly in the centre of the lesion. The lesion can be irregular or round in shape. Few of small abscesses can cluster to form large abscess which is known as **cluster sign**. On contrast enhanced CT, in arterial phase, the region around central hypodense area shows enhancement and the outer most layer also appears hypodense. This is the **double target sign**[42]**.** Also there can be wedge shaped enhancement is seen around the abscess, which is due to inflammatory changes in the portal vein due to abscess. This disappears in late arterial phase. In portal venous phase, thick ring like enhancement is noted in the region surrounding the hypodense centre.

Amoebic abscess

E. histolytica is the causative organism of amoebic liver abscess. These organisms reach the liver from the GIT via the portal vein. It's often difficult to differentiate from the pyogenic abscess. The clinical, epidemiological and serum antibody titres helps in its diagnosis. The CECT features includes rounded well defined hypo attenuating lesion (10-20 HU) with enhancing wall and edema surrounding it. The centre of the abscess cavity can show the septae, air bubbles, fluid debris level or haemorrhage[43].

Fungal abscess

Occurs in immunocompromised patients like AIDS, leukemia, bone marrow transplant. Microabscess show ring enhancement in arterial phase[44]. On equilibrium phase it appears hypodense.

2.HEPATIC INFARCTION

This condition is relatively rare as liver has dual blood supply from hepatic artery and portal vein. Hepatic infarction has various causes which includes shock, sepsis, emboli in rheumatic heart disease, bacterial endocarditis and metastatic tumor, arteriosclerosis, sickle cell anemia, eclampsia. Hepatic artery occlusion is commonly seen in liver transplant individuals who develop stenosis or thrombosis commonly at the anastomotic site between the donor and recipient artery.

CECT shows an ill-defined, wedge shaped hypoattenuating area and it is located peripherally without mass effect on adjacent structures[45]. There is

diminished enhancement of the lesion on contrast study. On MDCT can demonstrate the exact location of the intraluminal thrombus, stenosis or embolus.

3.BUDD CHIARI SYNDROME

The budd-chiari syndrome is a characterised by hepatic venous outflow obstruction at the level of hepatic vessels, the large hepatic veins, the IVC or the right atrium.

Factors that predispose to the development of buddchiari syndrome includes hypercoagulable states for eg. paroxysmal nocturnal hemoglobinuria, antiphospholipid antibody syndrome and inherited deficiencies of protein C, protein S and antithrombin III. Pregnancy, post partum period or it can be idiopathic. Obstruction of the hepatic venous outflow tract results in increased hepatic sinusoidal pressure if the consequent venous stasis and congestion leads to hypoxic damage to adjacent hepatic parenchymal cells.

In acute stages, the liver on CT appears enlarged and diffusely hypodense. These findings are attributed to hepatic congestion. Hepatic venous thrombosis is seen as a hypodense clot surrounded by enhancing vessel wall[46]. Normal or increased enhancement of caudate lobe is seen. Sub capsular enhancement may also be seen as these areas are drained by capsular veins. Patchy heterogeneous enhancement is seen in the rest of the liver due to diminished perfusion.

IV.DISEASES OF SPLEEN:

1.SPLENIC INFARCT

Splenic infarct occurs commonly as splenic arterial branches are end arteries with no inter-communication. Common causes include embolic disease, arteritis, splenic artery aneurysm, sickle cell disease and mass lesions.

On CT, infarcts are usually wedge shaped but may be irregular in contour. Splenic infarcts show no enhancement, but there may be peripheral enhancement of the capsule[47]. Lack of mass effect and perisplenic changes suggests infarct over abscess. Infarcts also shrinks over time. Chronic spelenic infarction leads to near complete calcification and atrophy of spleen.

2.SPLENIC ABSCESS

Splenic abscesses are uncommon being reported in 0.14 – 0.7%. Aerobic organisms account for 57% of splenic abscesses being caused by staphylococcus, E.coli and salmonella species.

On CT, bacterial abscess tend to be solitary and multi-loculated.Septations may be present and they may have enhancing capsule. Presence of gas within abscess is diagnostic. Mass effect on surrounding structures can be seen in splenic abscess.

Fungal abscesses are commonly seen in neutropenic patients, most commonly in acute leukaemia or those undergoing chemotherapy . An increased rise is reported due to aggressive use of chemotherapy[48].

On CT, fungal abscesses typically present as multiple, small micro abscesses. They usually measures about 5-10 mm in diameter. They often have low attenuation although a focus of high attenuation or a **wheel within a wheel pattern** is noticed.

3.SPONTANEOUS SPLENIC RUPTURE

Non traumatic splenic rupture is a rare condition.

Causes of spontaneous splenic rupture[49]:

Infections - Infectious mononucleosis, hepatitis, bacterial endocarditis, TB, syphilis, malaria.

Haematological - Lymphoma, leukemia, myeloma, hemophilia, anticoagulant therapy.

Metabolic - Amyloidosis, sarcoidosis, gaucher's disease.

Local - Pancreatitis, splenic vein thrombosis, metastasis.

At CECT, intra splenic hematoma manifests as a well defined lesion with decreased attenuation relative to normal splenic tissue. A subcapsular hematoma also has low attenuation but it is cresentic in shape and flattens the spleen subjacent to the capsule. The extent of injury to spleen can be grouped as (1) subcapsular hematoma which is cresentic and is closely located to splenic margin. (2) intra parenchymal hematoma which has irregular margins causing mass effect and splenic enlargement.

V.DISEASES OF BILIARY SYSTEM:

1.CHOLECYSTITIS

Gall stones are the most common cause of acute cholecystitis, seen in over 90% of cases. It results from persistent obstruction of cystic duct or gall bladder neck with distension of gall bladder and increased intra luminal pressure.

Although CT is not the imaging modality of choice for evaluation of acute gall bladder diseases, it may supplement the information provided by USG and it is helpful in excluding other causes of pain. It is also useful adjunct to USG, when the findings are equivocal or when complicated cholelithiasis is suspected. The CT features of acute calculus cholecystitis include gall stones, thickening of gall bladder wall[50], pericholecystic inflammatory change such as fluid collection and fat stranding, high attenuation bile, blurring of interface between gall bladder and liver and a transient increased in the attenuation of liver adjacent to GB[51]. Of all these, pericholecystic inflammatory changes is most specific. Only about 75% of stones of detected with CT.

The CT findings of acute cholecystitis are classified into major and minor criteria. Major criteria include calculi, mural thickening of GB, pericholecystic fluid and sub serosal edema. Minor criteria includes gallbladder distension and sludge. GB wall thickening is a non specific finding. Punctuate foci of contrast enhancement corresponding to enhancing vessels within GB wall is seen.

Acute acalculous cholecystitis

It occurs in critically ill patients. The risk factors being major cardiovascular diseases, cardiopulmonary bypass, autoimmune diseases, diabetes, bacterial and fungal sepsis and AIDS. Expect the absence of gall stones, CT findings are similar to calculus cholecystitis.

Gangrenous cholecystitis

It is a severe form of acute cholecystitis associated with vascular compromise and intra mural hemorrhage, necrosis and intramural abscess formation. It occurs due to stone impaction in cystic duct that leads to ischemic necrosis of the wall.

Emphysematous cholecystitis

It is a rare complication of acute cholecystitis. Vascular compromise of cystic artery causes proliferation of gas producing organisms. These organisms causes production and penetration of gas into GB wall. Clostridium welchii and E.coli are common organisms. It commonly occurs in diabetic and male patients.

CT has higher sensitivity than USG in identification of this complication. CT shows gas within the GB walls or the non dependent portion of the lumen[52]. There may be extension into pericholecystic soft tissues. It may also go in for abscess formation or perforation. Free intra peritoneal gas denotes GB perforation.

2.CHOLANGITIS

Bacterial infection of the bile ducts occurs mostly in patients with biliary obstruction caused by either stones or a tumor. Obstruction of the bile ducts cause bile stasis and predisposes to infection.

On CT, CBD dilatation is the most common finding. Pneumoblia can also be seen. Parenchymal changes are due to extension of inflammatory process into the periportal tissues and surrounding liver and also due to dilatation of peribiliary venous plexuses and increased arterial flow.

The presence of purulent bile is seen as increased attenuation of bile. Depending upon its calcium content, stones may also be depicted. The wall of bile ducts may be thickened concentrically and diffusely show dense contrast enhancement.

VI.DISEASES OF PANCREAS:

1.ACUTE PANCREATITIS

This is an acute inflammatory process involving the pancreas, most common causes being alcoholism, cholelithasis, followed by trauma, hyper-lipidemia, hypercalcemia, drug induced, tumors, and congenital anomalies.

Acute pancreatitis is broadly classified into acute interstitial pancreatitis and acute necrotizing pancreatitis[54].

Acute interstitial pancreatitis

This is often self limited with minimal organ impairment. Complications are limited and respond well to conservative treatment .Radiological investigations are indicated mainly to identify cause of pancreatitis[55]. Also when pancreatitis does not respond to treatment, CT is indicated with main aim of confirming the diagnosis, assessing severity, cause of pancreatitis and associated complications.

On CT pancreas appears minimally enlarged with low or heterogeneous attenuation of gland and it can have a shaggy contour[56]. Haziness of peripancreatic fat can be seen due to inflammation. Low attenuation areas in the peripancreatic region is due to extravasation of pancreatic fluid, fat necrosis and hemorrhage.

Acute necrotizing pancreatitis

This condition is the severe form of acute pancreatitis, characterized by severe inflammation associated with patchy or confluent areas of necrosis of pancreatic parenchyma.The revised ATLANTA classification[57] describes three forms of pancreatitis, (1) pancreatic parenchymal necrosis (2) peri-pancreatic necrosis (3) pancreatic necrosis with peri-pancreatic necrosis. Among them, peri-pancreatic necrosis is associated with better prognosis than parenchymal necrosis.

An initial non contrast enhanced CT is done to rule out alternative diagnosis. But, to assess the severity of pancreatitis, contrast enhanced CT is

done ideally 48 to 72 hours from the onset of pancreatitis. CECT is helpful to differentiate gland from the fluid collection and peri-pancreatic inflammatory tissue. Diminished or no contrast enhancement on CECT identifies areas of pancreatic necrosis and CT has a sensitivity of 100% to demonstrate extended pancreatic necrosis and sensitivity of 50% in demonstrating focal pancreatic necrosis.

The modified CT severity index is widely used to grade the severity of acute pancreatitis. It takes into consideration the CT grade, percentage of necrosis and extra pancreatic complications. This index has got a strong prognostic correlation and can predict length of hospital stay and morbidity.

Modified CT severity index

CT Grade	(Points)	Percentage Necrosis	(Points)	Extrapancreatic Complications	(Points)	Modified Severity Index = CT grade + percentage necrosis + extrapancreatic complications (points)
Normal pancreas	0	0	0	Pleural effusion, ascites, vascular complications, extrapancreatic parenchymal abnormalities, or gastrointestinal tract involvement	2	Mild (0-2)
Inflammation—pancreas and/or peripancreatic fat	2	≤30%	2			Moderate (4-6)
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4	>30%	4			Severe (8-10)

Acute fluid collections

These fluid collections which could be intra pancreatic or around the gland develop early in course of acute pancreatitis, depending upon presence of pancreatic necrosis and is seen to occur in about 40% of individuals acute fluid collection can be grouped as acute peri-pancreatic fluid collection (APFC) and acute necrotic collections (ANC) depending upon the presence of pancreatic necrosis[58] . These fluid collections lack a capsule and lie within the confines of anatomic space. The most common location being anterior pararenal space and lesser sac from these locations, the fluid can be seen extending into posterior pararenal space, mediastinum and solid organs like liver, spleen, kidneys.

On CT, fluid collections appear hypodense, with no capsule or wall. Most of these collections resolve spontaneously. The APFC collections that fail to resolve develop non - epithelised wall and become pseudocysts after about 4 weeks. ANC after 4 weeks termed as walled of necrosis.

Pseudocysts

Pseudocysts can be found anywhere in the abdomen and they can be seen in mediastinum and pelvis also. On CT, pseudocysts are round or oval shaped fluid collection with an enhancing wall[59]. Gas bubbles can be seen within them that indicate the presence of infection, fistula formation or due to cystotomy. There are several complications that occur due to pseudo cyst. Venous occlusion or stenosis can result in formation of varices. Arterial pseudo-aneurysm can occur due to enzymatic digestion of the arterial wall. High attenuation that is seen within the pseudo cyst is more likely due to the acute hemorrhage due to enzymatic digestion of vessels. Due to pressure effect by the pseudocyst gastro intestinal, biliary tract obstruction can be occur. Pseudocyst can rupture into peritoneal cavity or into the lumen of gastro intestinal tract, which can be indentified in CT. Fistulous communication into stomach or duodenum makes surgery unnecessary. But when fistula forms into the colon, bacterial contamination and infection can occur which needs surgical intervention.

Acute necrotic collection

Following the occurrence of necrotizing pancreatitis, the fluid collection containing necrotic pancreatic tissue and debris that develops in first four weeks is termed as walled off necrosis which could be sterile or infected

Walled off necrosis

Acute necrotic collections after 4 weeks or later can develop a thick non epithelised wall and this is known as walled off necrosis, that has replaced the term, pancreatic abscess. This contains non liquefied substance which are necrosed pancreatic tissue. On CT, they appear as thick walled hypodense collections that often contain air. The presence of air indicated the presence of infection or fistulous communication with gastrointestinal tract.

VII.DISEASES OF KIDNEY AND URETER:

1.RENAL AND URETERIC CALCULI

Since the advent of CT, it has become the gold standard for evaluating urolithiasis replacing radiography and urography.

Composition of calculi

Calcium stones (calcium oxalate mohohydrate, calcium oxalate dihydrate and calcium phosphate stones) form 70 – 80% of upper urinary tract stones. Struvite stones formed by magnesium ammonium phosphate accounts for 5-15% of stones. Uric acid stones form another 5 -10% of stones and form in acidic urine (pH 5.8). Other stones including cystine, xanthine and protein

matrix stones as well as stones formed as adverse effects of drugs (triamterene, indinavir) account for <5 % of all stones.

CT findings

NECT greater sensitivity (95-98%) and specificity (96-100%) in the diagnosis of urolithiasis[60]. CT also has the advantage of allowing other urinary and extraurinary abnormalities to be monitored. On CT, stones vary considerably in density, about 99% of calculi are visible on CT. The two radiolucent stones include indinavir stones and pure matrix stones. Which have soft tissue attenuation (15-30 HU) and likely to be missed in unenhanced CT. Dual energy CT assesses stone attenuation at two different kVp levels and helps in assessing the composition of calculi. MDCT helps in the identification of number, size location of calculi and presence of hydronephrosis. It also helps is assessment of stone fragility and composition with use of attenuation measurements and characterisation of internal structure. In IV contrast studies, the stones are seen as filling defects in contrast material filled pelvi-calyceal system and ureter.

Most direct CT signs for urolithiasis include visible stone within the lumen of ureter with proximal ureteral dilatation and normal distal calibre ureter. Ureteral stones are commonly lodged in proximal ureter (37%), distal uretrer (33%), mid ureter(7%) and UV junction (18%) and impacted ureteric stone show **soft tissue rim sign**[61]. The soft tissue rim represents edematous wall of ureter around calculus and has sensitivity of 50-77 % and specificity of 90- 100 %.The **comet tail sign**[62] is created by eccentric tapering of soft tissue

area adjacent to the calcification and is a reliable feature in the diagnosis of phleboliths. Another feature which differentiates phlebolith from calculus is the central lucent area seen in phleboliths in contrast with the opacified centers seen in the calculi.

The most reliable signs include hydronephrosis (83% sensitive, 94% specific) hydroureter[63] (90% sensitive, 93% specific) perinephric fluid (82% sensitive and 93% specific), periureteral edema and unilateral renal enlargement. Other less reliable findings of ureter calculi are absence of white renal pyramid , thickening of laterconal fascia and perinephric edema. Differences in renal parenchymal attenuation between obstructed and non obstructed kidneys is used as secondary sign of obstruction.

2.RENAL INFECTIONS

The diagnosis of renal infections are mostly made clinically. The main role of imaging is to identify whether there are underlying causes that predispose to infection like nephrolithiasis and to identify complications

Acute pyelonephritis

On CT, acute pyelonephritis presents with enlarged kidneys with perinephric fat stranding and thickening of renal fascia. On administration of contrast, striated nephrogram can be seen[64]. When the condition is diffuse, there can be decreased enhancement and delayed contrast excretion. When complications develop, hypo dense lesions with areas of water attenuation and bulging of renal surface with rounded margins and enhancing walls suggesting

presence of abscess[65] can be identified. Air pockets can also be seen within the abscess,.

Emphysematous pyelonephritis

This condition is life threatening, characterized by formation of gas within the renal parenchyma[66] .Diabetes being the most important predisposing factor. It can be grouped into two types .Type 1 – Destruction of more than 1/3rd of renal parenchyma and absence of renal or peri renal fluid collection. Type 2- Destruction of less than one third of renal parenchyma with formation of gas in collecting system and presence of renal and peri renal fluid collection. Type 2 has better prognosis[67].

Pyonephrosis

Accumulation of pus in obstructed pelvicalyceal system due to calculus, strictures, malignancy or developmental anomalies.

On CT the site and cause of obstruction can be demonstrated. Following administration of contrast, there is thickening of wall of renal pelvis, inflammatory changes in peri renal fat. Anterior to the pus in the dilated renal pelvis, contrast can be seen layered over it[68]. The presence of gas within the PCS in absence of instrumentation also indicates the presence of pyonephrosis.

3.RENAL INFARCTION

Renal infarction is caused by embolism, thrombosis, vasculitis, sickle cell disease or dissection of aorta. Among these causes thrombo-embolism constitute the most common cause.

On CT, renal infarct size depends on the occluded artery size. When the entire artery is occluded the entire kidney goes for infarction and appears diffusely hypodense, enlarged but reniform shape is maintained. In branch artery occlusion, it is seen as wedge shaped hypodensity with base towards the renal capsule and apex directed towards the hilum. On CECT, the hypodense area does not enhance but there can be a enhancing cortical rim due to preserved perfusion of outer rim of cortex by collateral vessels[69]. Sub capsular perinephric fluid collection or blood collection can be seen associated with thickening of renal fascia.

VIII.DISEASES OF VASCULAR SYSTEM:

1.AORTIC ANEURYSM RUPTURE

The risk of rupture of aortic aneurysm increases with increasing size of aneurysm, especially when diameter is more than 5.9 cm. Hence, surgery is advised for those with diameter more than 5.5 cm. CT is modality of choice for imaging aortic aneurysm and its complications[70]. Aneurysms can rupture into mediastinum, pleura, pericardium, airway, esophagus. The periaortic hematoma is seen with loss of aortic wall integrity, which is commonly

postero-lateral but can also be circumferential. Peri-aneurysmal fat stranding can be also be seen.

On CECT, contrast can be seen extravasating from the lumen of aorta into the site of rupture. CT can show hyper-dense crescent in the mural thrombus, which is an ominous sign suggesting impending rupture[71]. Another sign of impending rupture is **draped aorta sign**, where there is close apposition between aorta and spine which is due to deficient aortic wall and the aorta appears indistinct from the vertebral body and psoas muscle, with loss of peri – aortic fat planes. The direct signs of rupture of aortic aneurysm on CECT, include the presence of retroperitoneal hematoma or there can be extravasation of the intravenously administered contrast material[72].

2.AORTIC DISSECTION:

Aortic dissection potentially has a fatal outcome, which is in turn determined by type and extent of dissection. Dissection occurs as a result of spontaneous longitudinal separation of aortic intima by circulating blood. The blood filled space in media becomes the false lumen[73].

Aortic dissection can be classified as[74] :

Stand ford A: Which involves the ascending aorta extending to descending aorta, this requires urgent surgical intervention .

Stand ford B: Dissection involves descending aorta distal to left subclavian artery and treatment is mainly medical management.

On CECT, the intimal flap is seen in about 70% of cases, which separate true and false lumens. To differentiate true and false lumen, the signs that indicate false lumen include larger cross sectional area, **cobweb sign and beak sign**[75]. Cobweb sign is the slender linear hypo dense area due to residual media that have incompletely separated. Beak sign refers to wedge of hematoma that creates space for false lumen development. The true lumen can be seen to have continuity with undissected portion of aorta.

REVIEW OF EARLIER STUDIES BASED ON CT EVALUATION OF NON TRAUMATIC ACUTE ABDOMEN:

A study by rosen et al had been conducted with the aim of documenting the impact of abdominal CT performed on patients presenting to the emergency department with non traumatic acute abdominal pain. In this study the emergency department clinicians reported their most probable diagnosis, the level of certainty on a scale of 0-100% and the management plan of each of their patients before they order for abdominal CT. A total of 57 patients were enrolled in this prospective study. Specific protocol was used depending on the clinical indication of the patient. Non ionic IV contrast medium was used when required. The CT scans were obtained in 1.5-7mm interval spacing. The physicians level of diagnostic accuracy before and after performing CT scans were obtained for 48 patients and 9 patients had lost follow up and these information were not available for them. In the 48 patients studied, the level of certainty increased in 39 patients and remained unchanged in 6 patients and decreased in 3 patients. The difference in the level of certainty was assessed in

a five point scale with 5 being the maximum. For the 39 patients who had increase in levels of certainty, the mean increase was 2.0 points. Among the three patients who had decrease in certainty level, the mean decrease was 1.3 points. There was significant increase in the physician level of certainty after the performance of CT as the P value obtained was less than 0.0001.

The planned treatment before and after performance of CT was assessed in 55 patients. The management was altered in 33 of 55 patients. In 12 patients, hospital admission was planned but after CT the treatment plan allowed for patient to be managed at home. In two patients, the initial management was to send the patient home but after performance of CT, hospital based management was done. The diagnosis before and after the performance of CT was compared. In 27 patients the diagnosis before and after treatment coincided. In 30 patients there was discordance with the initial diagnosis. CT provided alternative diagnosis thus changing the management plan. Thus, the study proved that CT performed in emergency setting increases the probability of arriving at a correct diagnosis, raises the level of certainty in patients and helps to provide a specific diagnosis.

In another study by gore et al, the CT features of the commonly encountered acute abdominal conditions were described. The CT scans were obtained from the diaphragm to the pubic symphysis with a collimation of 5-7mm.The data were reconstructed at 3-7mm intervals depending upon the clinical indication, IV contrast media, oral and rectal contrast media were used when necessary depending upon the clinical indications.

Appendicitis was the most commonly encountered emergency. In few of those cases the clinical diagnosis was often atypical. The use of helical CT reduced the negative appendicectomy rate. The CT findings depicted the severity of inflammation. Appendix appeared distended with circumferential wall thickening with periappendiceal inflammation, calcified appendicolith, arrow head sign, caecal bar sign, focal caecal apical thickening sign can be present. The sensitivity and specificity of CT in the diagnosis of appendicitis is 90-100% to 83-97% respectively.

On CT, diverticulitis appeared as rounded outpouching in the paracolic region with the inflammatory changes at pericolic fat. The presence of tethered saw tooth configuration with engorged vasa recta pointed towards the diagnosis of acute diverticulitis. CT helped to differentiate diverticulitis from appendicitis, typhilitis, epiploic appendagitis.

CT in small bowel obstruction helped to identify the level, cause of obstruction. It could differentiate closed and open loop obstruction. CT had a sensitivity of 90-96% and specificity of 91-96%

The features seen in CT in bowel ischaemia depends upon the cause, chronicity, severity of intestinal ischemia, mural thickening was the most common finding. The wall of the bowel had halo or target appearance, focal pneumatosis was also seen. Ischemic colitis presented with irregular scalloped margins due to the presence of edema. Epiploic appendagitis on CT appeared as a fat attenuation mass with hyperdense central focus a hyperdense rim.

On CT, mesenteric adenitis was diagnosed when the lymph nodes are more than 5mm in short axis diameter with surrounding inflammation in the mesentery. Abscess formation and bowel obstruction form the emergencies in crohns disease. MDCT effectively identifies bowel wall thickening and state of diseased bowel. The site of location of abscess and image guided drainage can be effectively assessed with CT. In patients with ulcerative colitis the bowel wall status, toxic megacolon and perforation could be identified.

Perforation was diagnosed in CT when X-ray could not. Loculated air or fluid level, focal parietal peritoneal enhancement was seen.

Intra-abdominal abscess appeared as low attenuation centre with enhancing with displacement of the surrounding structures.

The most sensitive finding in acute cholecystitis was GB wall thickening of more than 3mm with enhancement of the wall on contrast administration. CT has a sensitivity of 88% and specificity of 97% in diagnosing acute cholecystitis.

On CT, acute pancreatitis was seen as enlarged edematous gland with peripancreatic fat stranding with irregular contour. In advanced cases intrapancreatic fluid collection can be seen. In necrotizing pancreatitis, the enlarged gland contains necrosed pancreas which was seen as high attenuation fluid collection. The severity of necrosis was found to correlate with the mortality. In the absence of necrosis there was no mortality and 6% morbidity.

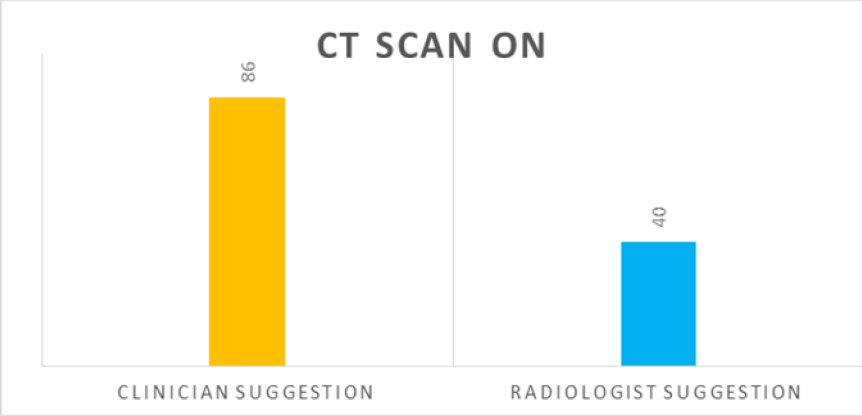
When necrosis was less than 30%, there was no mortality and 40% morbidity. When the necrosis was more than 50%, patients had 75-100% morbidity and 11-25% mortality.

CT angiogram with >3ml/sec infusion of contrast agent was used to image the aortic dissection. The intimal flaps, true and false lumen would be depicted. Rupture of aortic aneurysm resulted in formation of retroperitoneal hematoma which was seen on CECT as contrast extravasation.

OBSERVATION

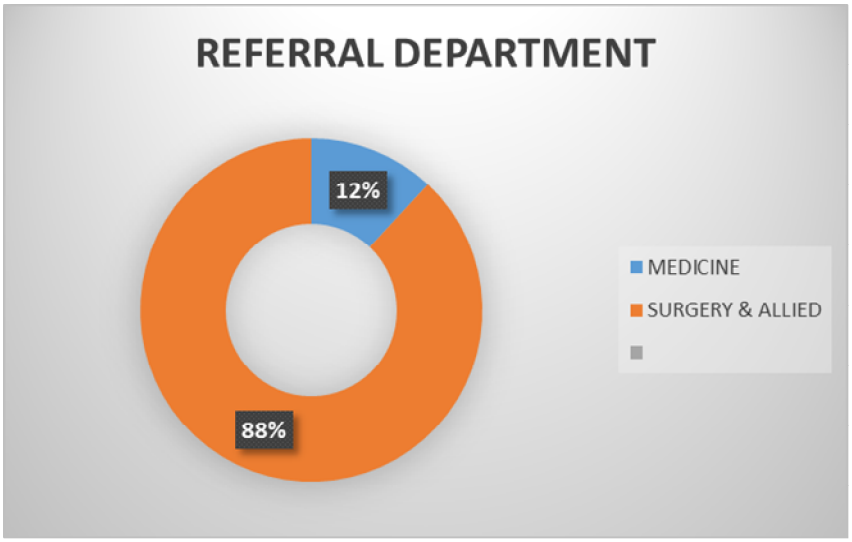
The present prospective study was done on 126 patients who presented to the emergency department with acute abdominal pain. CT abdomen and pelvis was done for those patients in whom ultrasound could not yield a definitive diagnosis or when the clinician had referred the patients for CT abdomen and pelvis to obtain further information regarding the diagnosis.

CHART-I: CT SUGGESTED BY CLINICIAN AND RADIOLOGIST



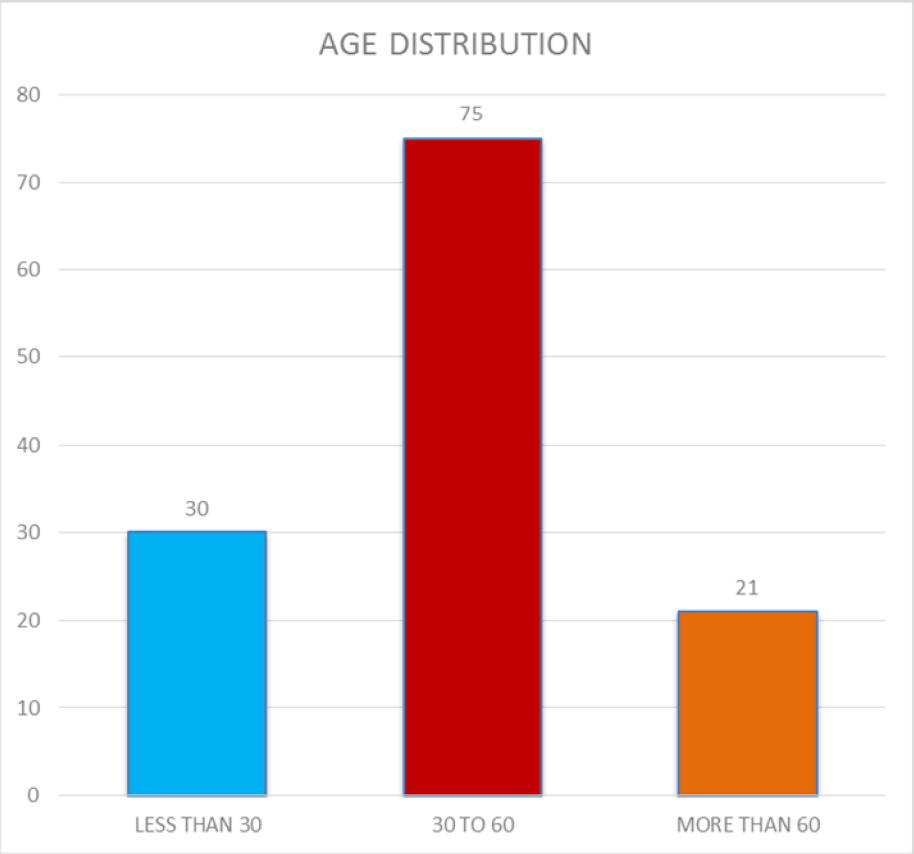
Majority of the patients, that is 111patients were referred from the surgical and surgical allied emergency departments and 15 patients were referred from the emergency medicine ward.

CHART – II: REFFERAL DEPARTMENT



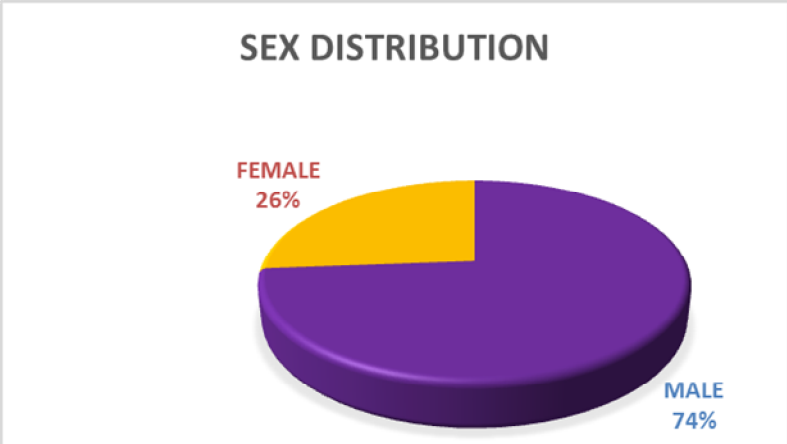
Among the 126 patients, 30 patients belonged to age group less than 30 years, 75 patients belonged to 30-60 years age group and 21 patients belonged to age more than 60 years.

CHART-III: AGE DISTRIBUTION



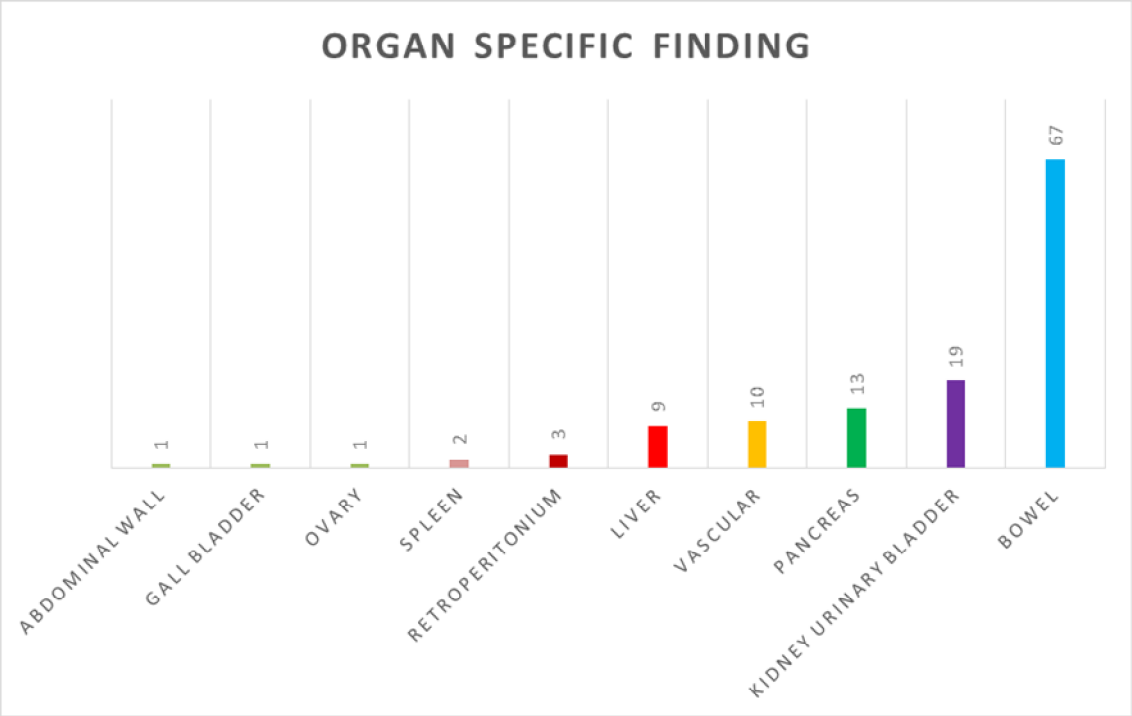
Among 126 patients in our study, majority of the patients were males. 93 (74%) patients were male and 33 (26%) patients were female.

CHART-IV: SEX DISTRIBUTION



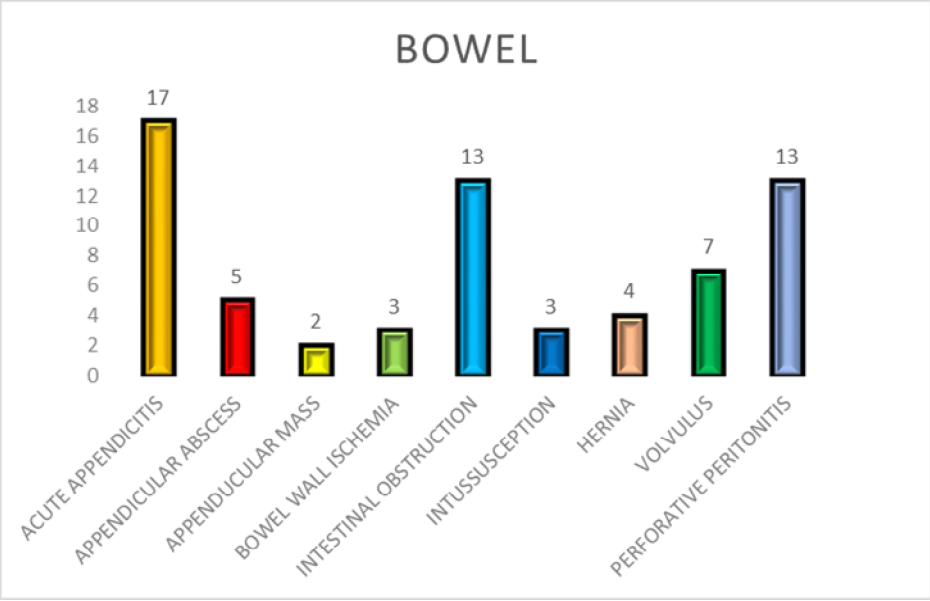
CT abdomen and pelvis was done in 126 patients and the various diagnosis obtained were tabulated and grouped into organ specific diagnosis. In our study, diseases pertaining to bowel scored the highest followed by kidney and ureter and the pancreas followed by other abdominal organs.

CHART-V: ORGAN SPECIFIC FINDING



Among the 67 patients with disorders of the gastrointestinal tract, acute appendicitis was the most frequently encountered. Acute appendicitis was the most commonly observed surgical emergency in our study, which was observed in 17 patients. Perforative peritonitis constituted the next most commonly observed pathology in this group of patients.

CHART –VI: DISTRIBUTION OF DISEASES PERTAINING TO BOWEL



The ultra-sonographic features of the 126 patients were compared with the findings obtained in computed tomography. Compared to USG, CT was better in achieving a specific diagnosis. CT could pick up 8 cases of appendicitis which were not picked up in USG. Similarly, CT could pick 3 cases of bowel perforation, 10 cases of small bowel obstruction, 3 cases of bowel ischemia, 1 case of aortic aneurysm, 10 cases of intra-abdominal abscess, 7 cases of pyelonephritis, 5 cases of renal and ureteric calculi, 5 cases of volvulus and 8 cases of acute pancreatitis which were not clearly depicted in USG and CT helped in providing the appropriate treatment.

TABLE-I: ADDITIONAL CASES DIAGNOSED BY CT

	WITH USG	AFTER CT	DIFFERENCE
ABSCESS(ALL)	15	25	+10
AORTIC ANEURYSM	3	4	+1
APPENDICITS	9	17	+8
BOWEL PERFORATION	10	13	+3
CHOLECYSTITIS	1	1	0
SMALL BOWEL ISCHAEMIA	0	3	+3
INTESTINAL OBSTRUCTION	10	13	+3
RENAL & URETERIC CALCULUS	2	7	+5
PYELONEPHRITIS	4	11	+7
ACUTE PANCREATITIS	4	12	+8
VOLVULUS	2	7	+5

CHART –VII: DIAGNOSIS BEFORE AND AFTER CT

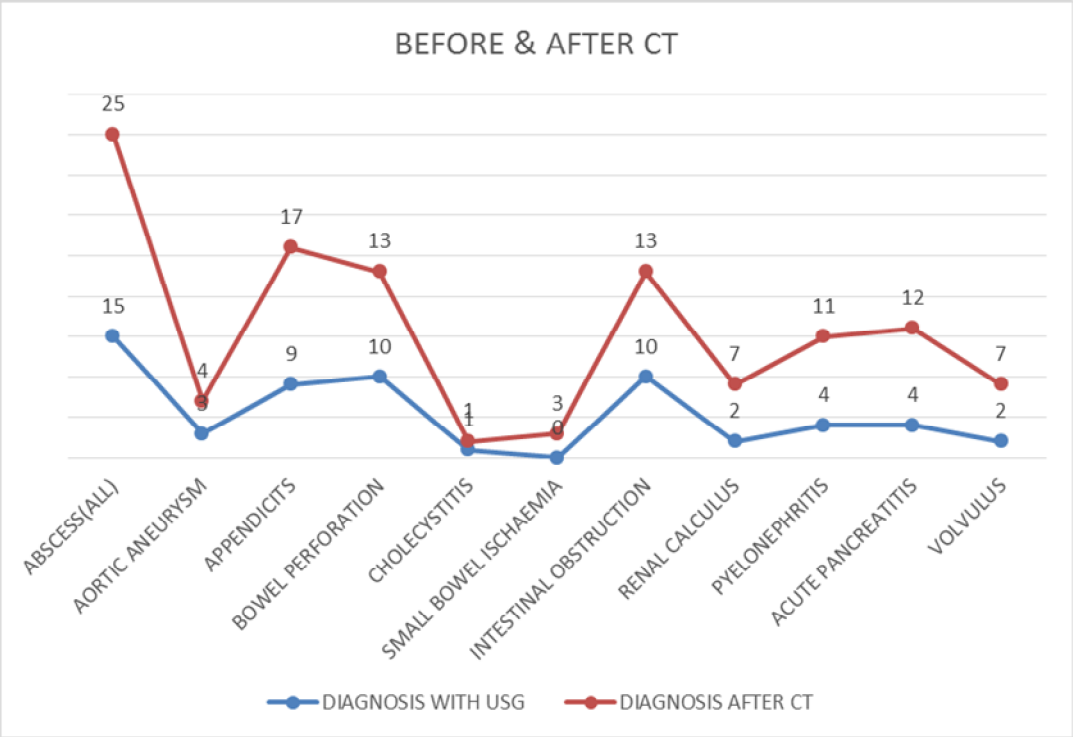


TABLE – II DISEASE SPECIFIC DISTRIBUTION:

DISEASE SPECIFIC	
DIAGNOSIS IN CT	**NO OF CASES**
LIVER ABSCESS	9
SPLENIC ABSCESS	2
SPLENIC RUPTURE	1
ABDOMINAL WALL HEMATOMA	1
EMPYSEMATOUS CHOLECYSTITIS	1
TORSION OVARY	1
RETRO PERITONEAL ABCESS	2
ILIO PSOAS ABCESS	1
CALCULUS	7
PYELONEPHRITIS	11
SUBPHRENIC ABSCESS	1
ACUTE APPENDICITIS	17
APPENDICULAR ABSCESS	5
APPENDIUCULAR MASS	2
BOWEL WALL ISCHEMIA	3
INTESTINAL OBSTRUCTION	13
INTUSSUCEPTION	3
VOLVULUS	7
PERFORATIVE PERITONITIS	13
ACUTE PANCREATITIS	10
PANCREATIC ABSCESS	1
PANCREATIC NECROTIC COLLECTION	1
AORTIC ANEURYSM	2
AORTIC DISSECTION	2
SMV THROMBUS	2
SMA THROMBUS	2
SMA SYNDROME	1
RENAL ARTERY THROMBUS	1
OTHERS	4

On follow up of these patients, 93 patients were operated and 33 patients were managed conservatively. In 13 patients, surgery was planned before performance of CT. After CT was performed, there was a change in diagnosis and these patients were put on conservative treatment. In five patients who were planned to be managed conservatively, after CT was performed there was a change in the final diagnosis and surgery was performed.

TABLE –III: DEPICTING THE CHANGE IN SURGICAL MANAGEMENT AFTER CT

	PLANNED FOR SURGERY
BEFORE CT	106
AFTER CT	93

CHART –VIII: CHANGE IN SURGICAL MANAGEMENT AFTER CT

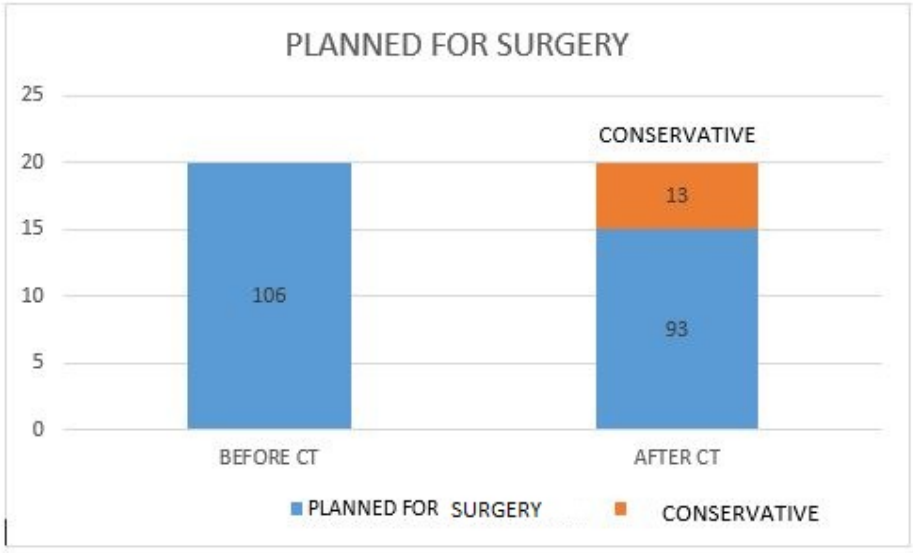
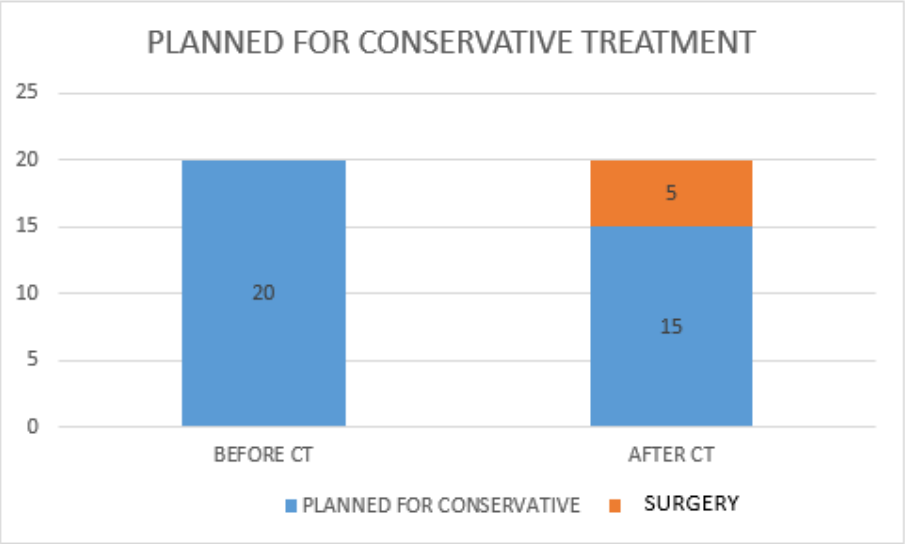


TABLE –IV: DEPICTING THE CHANGE IN CONSERAVTIVE MANAGEMENT AFTER CT

	PLANNED FOR CONSERAVTIVE TREATMENT
BEFORE CT	20
AFTER CT	15

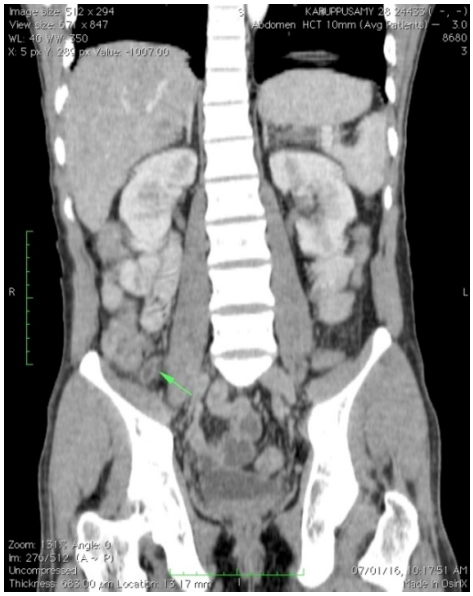
CHART –IX: CHANGE IN CONSERAVTIVE MANAGEMENT AFTER CT



REPRESENTATIVE CASE

REPRESENTATIVE CASE - 1 : ACUTE APPENDICITIS

A 28 year old male patient presented with right lower quadrant pain, fever and vomiting for 3 days. CECT findings of the patient



FINDINGS :

The appendix inflamed and thickened. Maximum diameter measuring 1cm in size. Peri- appendicular fat stranding noted. No obvious mass /abscess formation.

REPRESENTATIVE CASE - 2 : APPENDICULAR ABSCESS

A 72 year old male patient presented with fever, vomiting, abdominal pain for past 10 days . CECT findings of the patient,

FINDINGS :

There is loculated collection with irregular enhancing walls noted in right iliac fossa.

REPRESENTATIVE CASE - 3 : EPIPLOIC APPENDAGITIS

A 36 year old patient presented with left lower abdominal pain for 5 days. CECT done which revealed the following findings,

FINDINGS :

Pericolonic oval shaped lesion with fat attenuation with periappendageal fat stranding with surrounding hyperdense rim which represents parietal peritoneal thickening. There is a central high attenuation dot within it that represents thrombosed vessel

REPRESENTATIVE CASE - 4: GASTRO-JEJUNAL INTUSSUSCEPTION

50 year old male with past history of gastro-jejunostomy with truncal vagotomy presented with epigastric pain, abdominal distension and vomiting. CECT was done for the patient.

FINDINGS:

Grossly distended stomach with jejunal loops seen prolapsing into the stomach with central fat density area suggesting the mesenteric fat which had intussuscepted into the stomach along with the jejunal loops.

REPRESENTATIVE CASE – 5 : INTUSSUSCEPTION

31 year old female presented with colicky abdominal pain, vomiting, abdominal distension and obstipation for 3 days. CECT abdomen and pelvis was done.

FINDINGS;

Ileocolic and ileo-ileal intussusception noted with polyps as the lead point. Multiple mildy enhancing polyps seen in the jejunal and ileal loops. Upon follow up of the patient, biopsy of the polyps proved them to be hamartomatous polyps and thus a diagnosis of peutz jegher's syndrome with multiple intussusception was made.

REPRESENTATIVE CASE - 6 : MID GUT VOLVULUS

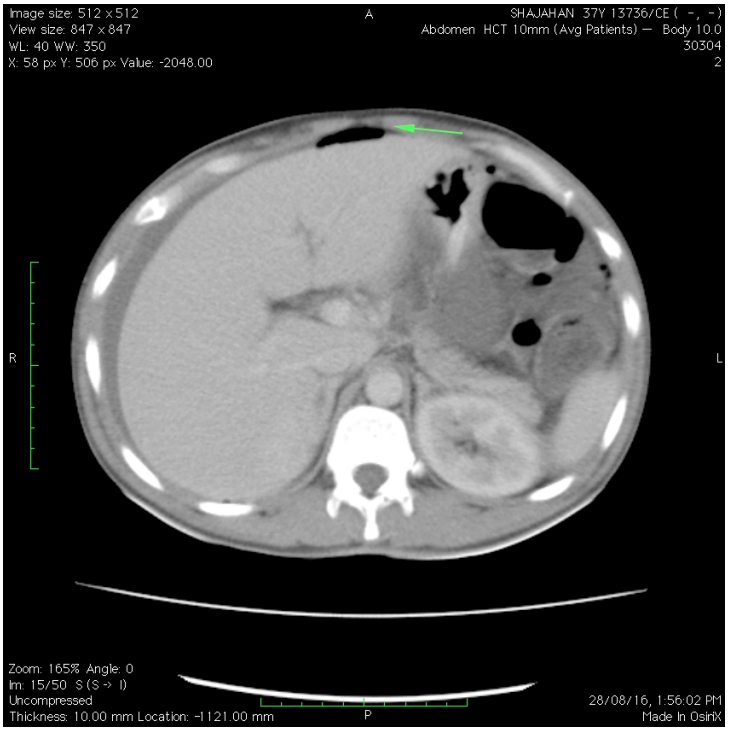
A 27 years old female patient presented with abdominal pain, distension, vomiting and constipation for one day. CECT of abdomen and pelvis done

FINDINGS :

There is twisting of mesentry along with bowel loops with proximal dilatation of stomach and small bowel loops seen. The SMA and SMV axis is altered.

REPRESENTATIVE CASE - 7 : PERFORATIVE PERITONITIS

A 37 years old male patient presented with acute onset of abdominal pain for one day. CECT findings of the patient



FINDINGS :

Free fluid seen in abdomen and pelvis with pockets of free intra-peritoneal air seen. Enhancing smooth peritoneal thickening noted.

REPRESENTATIVE CASE - 8 : SMALL BOWEL OBSTRUCTIUON

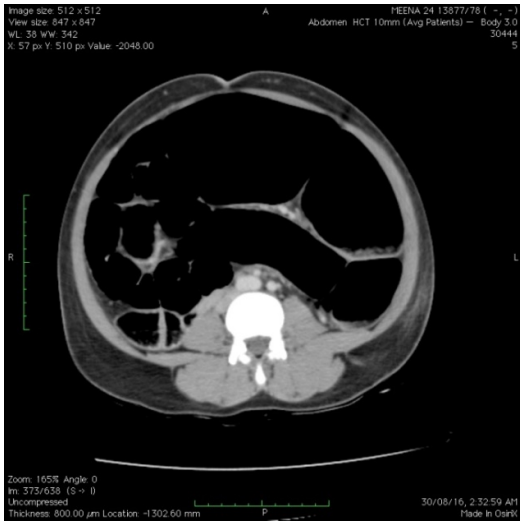
A 16 year old male patient presented with history of abdominal pain and distension, vomiting and obstipation for 3 days.

FINDINGS :

CECT abdomen and pelvis shows, dilated small bowel loops upto the level of distal ileum with multiple air fluid levels. The colonic bowel loops appears collapsed. Transition zone was seen at the terminal ileum. Per-operatively stricture was found at the transition zone .

REPRESENTATIVE CASE – 9 : SIGMOID VOLVULUS

A 24 years old female patient presented with acute onset of abdominal pain for one day.

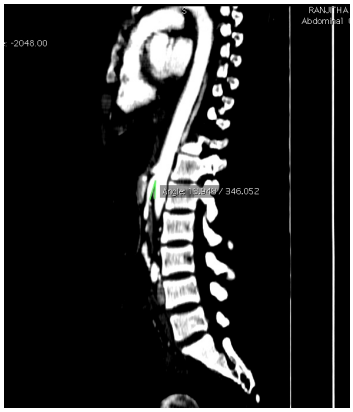


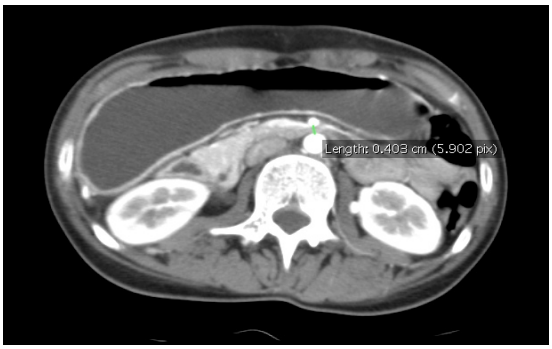
FINDINGS :

CECT shows the abnormally positioned and grossly dilated, gas filled sigmoid colon with swirling of mesentery and vessels seen in the pelvis, which is the site of twist. Coronal reformation of CT, shows the U-shaped closed loop appearance of dilated sigmoid colon with three line sign of opposed walls of dilated bowel loops.

REPRESENTATIVE CASE -10: SMA SYNDROME

20 year old female presented with abdominal pain and distension for 2 days.



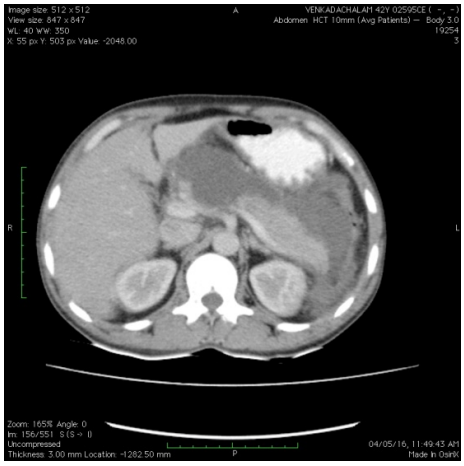


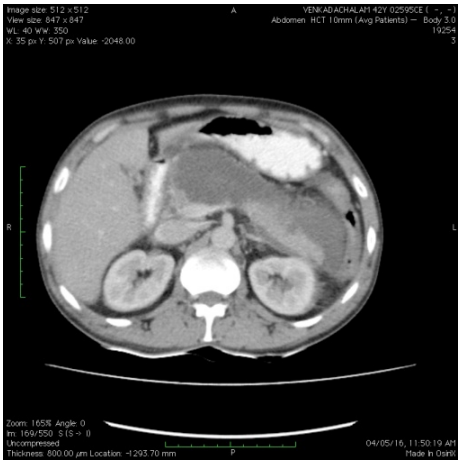
FINDINGS;

CT showed grossly dilated stomach and duodenum upto its third part. The third part of duodenum shows abrupt narrowing at the level of crossing of SMA.CT Abdominal Angiography revealed reduced Aorta-SMA angle measuring 13 degrees and reduced Aorta-SMA distance that measured 4mm

REPRESENTATIVE CASE - 11 : ACUTE NECROTIZING PANCREATITIS

A 42 year old male patient presented with complaints of abdominal pain and distension and vomiting for 5 days. CECT abdomen and pelvis done.





FINDINGS :

Pancreas enlarged in size, necrotic areas noted within the pancreas and shows heterogeneous enhancement in contrast study. There is acute necrotic collection noted in peri – pancreatic region with thickening of gerota's fascia and lateral conal fascia.

DISCUSSION

The main aim of our study was to illustrate the importance of CT in obtaining a specific diagnosis in patients with non-traumatic acute abdomen. Initially ultrasound was performed for these patients and CT was performed when ultrasound was inconclusive or when the clinician wanted additional information. The diagnosis obtained by ultrasonography and that obtained by CT were compared with the per -operative or final diagnosis at discharge. In our study, CT was found to be better than ultrasonography in finalizing the diagnosis. Similarly the impact of CT on the management of these patients was assessed.

In a study conducted by Rosen et al on 57 patients presenting with non traumatic acute abdomen it was stated that abdominal CT could change the planned treatment in 33 patients. The planned treatment based upon the clinical diagnosis was hospital based management in 42 patients. But after performing CT, a total of 32 patients were only admitted, thus CT could avert 10 among these 42 admissions. In this study after performing CT, 2 patients who were initially planned to be sent home were admitted. Additionally 6 patients who were planned on conservative treatment, underwent immediate surgery after performing CT.

Our study correlated with the study done by Rosen et al in proving that CT could provide the appropriate management for these patients . In our study the management was changed in 18 patients. 13 patients who were planned for immediate surgery, after performing CT, the management of these patients was

changed to conservative treatment. Similiarly 5 patients who were planned to be kept on observation, after performing CT were operated immediately.

In our study the diagnosis obtained through ultrasound and CT were compared. CT scored over ultrasound in diagnosing and detecting the complications of several conditions such as acute appendicitis, hollow viscus perforation, volvulus, pancreatitis, pyelonephritis, uretertic stones and abdominal vascular pathology.

Among the total of 17 cases of acute appendicitis, ultrasound could diagnose only 9 cases but CT could diagnose additional 8 cases , which were not suspected in ultrasonography. Appendix when especially retro-caecal in position is difficult to visualize, because of the caecal gas shadows. These cases could be diagnosed by the help of CT. Early cases of acute appendicitis which were minimally distended and measuring about 5-6 mm diameter could be picked up by CT. The complications such as perforated appendix, appendicular abscess, intra peritoneal abscess were better detected through CT.

A total of 13 cases of hollow viscus perforation were diagnosed. 10 cases were diagnosed in ultrasonography. Additional of three cases of hollow viscus perforation could be detected by CT. Tiny pockets of free intra-peritoneal air may go undetected in ultrasonography, but CT could readily detect the same.

CT detected 7 cases of volvulus of which only 2 cases were diagnosed by ultrasonography. The 2 cases of volvulus that were diagnosed in

ultrasonography were mesenteric and mid gut volvulus. The other 5 cases of volvulus which could not be diagnosed in ultrasonography were cases of sigmoid volvulus. It was due to the extensively dilated gas filled large bowel loops preventing the penetration of ultrasound beam. In abdominal radiography in these 5 cases there was a strong suspicion of sigmoid volvulus. Thus CT proved to be a better imaging modality in in confirming a diagnosis of volvulus.

Three cases of bowel ischemia which were not diagnosed by ultrasound were detected in CT. In one of these, pneumatosis was picked up in the ileal loops and in the other two cases bowel wall thickening and intramural hemorrhage was identified in CT which could not be made out in ultrasonography.

Only one case of emphysematous cholecystitis was referred for CT. The main reason is higher sensitivity of ultrasonography in diagnosing the disorders of gall bladder and biliary system. Hence, CT was not essential for further evaluation.

A total of 12 cases of pancreatitis were included in our study. Ultrasound had missed 8 cases and it could diagnose only 4 cases. CT is superior in diagnosing acute pancreatitis and its complications. In ultrasonography, it is difficult to diagnose pancreatitis because of bowel gas and obesity. But, CT could overcome these limitations of ultrasonography. The complications of pancreatitis could be better detected in CT. The presence of pancreatic and peri- pancreatic fluid collections, pancreatic necrosis, pancreatic

abscess, pseudo-cyst and vascular complications could be better appreciated in CT. The CT severity index could be determined which helped in predicting the prognosis. The presence of peri-pancreatic fat stranding, loss of normal lobular contour of the pancreatic borders helped to diagnose early cases of acute interstitial pancreatitis which could not be made out in ultrasound. In our study, there was a case of pseudo-cyst of pancreas with cysto- gastric fistula. Ultrasound showed only the presence of pseudo-cyst in pancreas, but CT performed after administration of oral and IV contrast revealed the presence of fistulous communication between the cyst and stomach, thus proving that CT was superior in diagnosing and detecting the complications of acute pancreatitis.

Ureteric calculi were better detected in CT compared to ultrasound. In these cases, ultrasound only showed the presence of hydro-uretero-nephrosis because the mid and distal ureter tracing was poor due to the presence of bowel gas shadows. CT could detect the exact location, size of calculus and the severity of obstruction caused by it. CT is superior to USG to detect pyelonephritis, emphysematous pyelonephritis and their complications.

Vascular pathologies like SMA thrombus, SMV thrombus, SMA syndrome, renal artery thrombosis, aortic aneurysm and aortic dissection and their complications were better detected in CT. In one patient USG made the diagnosis was grossly dilated stomach and duodenum. After CECT was performed it was seen that the AMA angle was severely reduced causing

compression on third part of duodenum with no evidence of mass lesion. Thus the diagnosis of SMA syndrome was made in CT.

In the study conducted by Rosen et al , among the 57 patients, complete follow up could be done for 44 patients. CT could yield correct diagnosis in 41 patients. False positive diagnosis was made in 2 patients, in one patient CT showed thickening of transverse and descending colon but colonoscopy showed the presence of only lymphoid aggregates. In another case inflammatory changes were seen around the appendix and the case was diagnosed to as acute appendicitis but per - operative finding revealed normal appendix. One false negative diagnosis was made in a patient with right lower quadrant pain. CT revealed a normally looking appendix but per operatively appendix was inflamed, suggestive of appendicitis.

Similarly, the 126 cases in our study were followed up. The correct diagnosis was obtained in 125 cases. In our study, CT provided a false positive diagnosis in one patient. CT showed the presence of diverticuli with probable presence of rent in the diverticulum in the region of hepatic flexure. But per operatively, the colon appeared normal. Another was a case of small bowel obstruction at the level of distal ileum but the cause of obstruction could not be diagnosed in CT. Per-operatively, there was fallopian tube seen winding around the distal ileum which could not be diagnosed in CT.

In our study, it was proved that CT could provide timely and correct diagnosis in 125 patients among the 126 cases. In a few patients there was dis-cordance in the clinical diagnosis and CT diagnosis. On follow up of these

patients, the diagnosis made in CT was found to be correct and the previously planned management was changed. For example, in one case there was clinical suspicion of right ureteric colic when CECT was done, it showed presence of inflamed appendix apart from the right ureteric calculus. The patient was operated and the per-operative finding revealed inflamed appendix. Another case referred as left ureteric colic was diagnosed as epiploic appendagitis in CT and the patient was treated conservatively.

The disadvantage of CT is the cost and the radiation exposure. But CT provides a timely diagnosis and reduces the hospital stay and morbidity. Hence early and timely use of CT proved to be cost effective and decreases patient morbidity.

CONCLUSION

Acute abdominal pain is a common presenting symptom in the emergency department. Pain being a subjective symptom and the spectrum of causes of acute abdominal pain being broad, imaging plays a pivotal role in diagnosing the cause of acute abdominal pain. Making an appropriate diagnosis is essential in planning the appropriate management and reducing morbidity and mortality.

Though radiography is widely available, its use is limited mainly for hollow-viscus perforation and intestinal obstruction. USG can be inconclusive in the presence of extensive bowel gas or abdominal fat which would prevent adequate visualization of abdominal organs.

In our study it has been proved that CT helps in arriving at an accurate diagnosis. The associated complications of the underlying disease can also be determined with CT which helps in predicting the prognosis. CT can effectively guide the clinician regarding the management. It helps to determine who need surgery and who do not. Hence CT can be considered as the primary imaging with the exception of acute cholecystitis in which USG proved highly sensitive in the diagnosis.

Despite the small risk of radiation and the slightly increased cost, prompt utilization of CT in investigating cases of acute abdomen gives more accurate diagnosis and leads to better decision making regarding management, thus improving outcomes.

SUMMARY

The purpose of the study is to evaluate the importance of CT in diagnosing non- traumatic acute abdomen and to describe the spectrum of CT findings in acute abdomen.

Acute abdominal pain is a common presentation in emergency department. The causes are renal colic, small bowel obstruction, acute appendicitis, acute cholecystitis, pancreatitis, peritonitis, abscess, diverticulitis, bowel perforation ,bowel ischaemia , etc. While clinical examination and laboratory investigation may help to narrow down the probable diagnosis, imaging examination is absolutely needed to establish the correct diagnosis.

CT is more accurate than ultrasonogram and plain X-Ray. Both sensitivity and specificity of contrast enhanced CT is better than ultrasonogram. In a significant number of patients clinical management (surgery vs conservative) is significantly altered due to the findings detected in contrast enhanced CT.

Thus CT can be advocated both as a primary diagnostic modality or as a valuable adjunct to preliminary ultrasound whenever evaluation of acute abdomen is needed in the non – pregnant adult patient with normal renal function.

CONSENT FORM

Yourself Mr/Mrs/Ms ....., aged …… years , S/o / D/o / W/o……..... residing at ……are being asked to be a participant in the research study titled **"MDCT EVALUATION OF NON TRAUMATIC ACUTE ABDOMEN"** in Coimbatore Medical College and Hospital, Coimbatore, conducted by **Dr.BALAMURUGAN.P.P.,**one of the Post Graduate student in the department of Radio-diagnosis, Coimbatore Medical College and Hospital. You satisfy eligibility as per the inclusion criteria. You can ask any queries you may have before agreeing to participate.

TOPIC OF THE RESEARCH:

MDCT Evaluation of Non-Traumatic Acute Abdomen

PURPOSE OF RESEARCH:

The purpose of the study is to document the importance of CT to diagnose non traumatic acute abdomen and describe the spectrum of causes of acute abdomen.

PROCEDURES INVOLVED IN THE STUDY

CT Abdomen and pelvis axial section taken from diaphragm to beneath symphysis pubis.

Multiplanar reconstruction done at the intervals of 3-7 mm by using maximum intensity projection, minimum intensity projection and volume rendering.

Iodinated I V contrast routinely used except in patients suffering from medical renal disease and known anaphylaxis to medications.

Oral and Rectal contrast used whenever necessary.

I V Contrast -IOHEXOL (Omnipaque) 350 mg iodine /ml.

Dose -1.75 ml /kg (Avg -90 to 100 ml) by using power injector through I V cannula (18 Gauge)

Rate of administration - 2ml /sec.

Initially CT abdomen and pelvis axial section plain taken, followed by contrast.

DECLINING FROM PARTICIPATION:

You are hereby made aware that participation in this study is purely voluntary and honorary, and that you have all the rights to decline from participating in it.

PRIVACY AND CONFIDENTIALITY:

Privacy of individuals will be respected and any information provided will be kept confidential.

AUTHORISATION TO PUBLISH RESULTS:

Results of the study may be published for scientific purposes and/or presented to scientific groups; however you will not be identified.

STATEMENT OF CONSENT:

I ..... voluntarily give my consent to participate in this study being conducted by Dr. **BALAMURUGAN.P.P.**I have read the consent form/it has been read to me. The study has been fully explained to me and I understand that I am entitled to explanations regarding the study as and when necessary.

.....

Signature /Left thumb impression of patient/legal guardian of the child

Station : Coimbatore

Date :

.....

Signature / Left Thumb Impression and Name of witness

Coimbatore :

Date :

BIBILOGRAPHY

1. Terasawa T, Blackmore CC, Bent S, et al: Systematic review: computed tomography and ultrasonography to detect acute appendicitis in adults and adolescents. Ann Intern Med 2004; 141:537-546.
2. Daly CP, Cohan RH, Francis IR, Caoili EM, Ellis JH, Nan B. Incidence of acute appendicitis in patients with equivocal CT findings. AJR Am J Roentgenol 2005;184:1813– 1820
3. Fukuya T, Hawes DR, Lu CC, et al: CT diagnosis of small-bowel obstruction: efficacy in 60 patients. AJR Am J Roentgenol 1992; 158:765-769.discussion 771-772
4. Lazarus DE, Slywotsky C, Bennett GL, et al: Frequency and relevance of the "small-bowel feces" sign on CT in patients with small-bowel obstruction. AJR Am J Roentgenol 2004; 183:1361-1366.
5. Ha HK: CT in the early detection of strangulation in intestinal obstruction. Semin Ultrasound CT MR 1995; 16:141-150.
6. Federle MP, Chun G, Jeffrey RB, et al: Computed tomographic findings in bowel infarction. AJR Am J Roentgenol 1984; 142:91-95.
7. Ha HK, Kim JS, Lee MS, et al: Differentiation of simple and strangulated small-bowel obstructions: Usefulness of known CT criteria. Radiology 1997; 204:507-512.
8. AgrawalGA, Johnson PT, Fishman EK. Multidetector row CT of superior mesenteric artery syndrome. J ClinGastroenterol2007;41(1):62–65.

9. Na-ChiangMai W, Pojchamarnwiputh S, Le-rtprasertsuke N, Chitapanarux T. CT findings of tuberculous peritonitis. Singapore Med J 2008; 49:488–491
10. Kim SW, Kim HC, Yang DM. Perforated tumours in the gastrointestinal tract: CT findings and clinical implications. Br J Radiol 2012; 85:1307–1313.
11. Pradel JA, Adell JF, Taourel P, et al: Acute colonic diverticulitis: Prospective comparative evaluation with US and CT. Radiology 1997; 205:503-512.
12. SinghAK, Gervais DA, Hahn PF, Rhea J, Mueller PR. CT of acute appendagitis. AJR Am J Roentgenol2004; 183: 1303–1307.
13. McClure MJ, KhaliliK, SarrazinJ, HanbidgeA. Radiological features of epiploicappendagitis and segmental omental infarction. ClinRadiol2001;56(10): 819–827.
14. WiesnerW, Kaplan V, BongartzG. Omental infarction associated with right-sided heart failure. EurRadiol2000;10(7):1130–1132.
15. Pear BL. Pneumatosisintestinalis: a review. Radiology. 1998;207:13–19.
16. Kernagis LY, Levine MS, Jacobs JE. Pneumatosisintestinalis in patients with ischemia: correlation of CT findings with viability of the bowel. AJR Am J Roentgenol. 2003;180:733–736.
17. GayerG, Apter S, Hofmann C, et al. Intussusception in adults: CT diagnosis. Clin Radiol1998; 53: 53–57
18. Bapaye M, Kolte S, Pai K, et al. Jejunogastric Intussusception presenting with outlet obstruction. Indian J Gastroenterol 2003;22:31-2.

19. Bakhash K, Igbinovia A, Egere JU, Ali A, Butt MS, Rehan AM. Jejunogastric intussusception: A rare cause of life threatening hematemesis. Annals Saudi Med 1997;17:328-30.
20. Oldenburg WA, Lau LL, Rodenberg TJ, et al: Acute mesenteric ischemia: A clinical review. Arch Intern Med 2004; 164:1054-1062.
21. Bradbury MS, Kavanagh PV, Bechtold RE, et al: Mesenteric venous thrombosis: Diagnosis and noninvasive imaging. Radiographics 2002; 22:527-541.
22. Mathew D.lucianiA.Internal abdominal herniations.AJR AMJ Roentgenol 2004;183:397-404
23. Aguirre DA,Santosa AC, Casola G, SIrlinCB.Abdominalwaal hernias :imaging features, complications and diagnostic pitfalls at multidetector row CT.Radiographics 2005;25:1501-1520
24. Miller PA;Mezwa DG , FeczhoPJ,JafriZH,madrazoBL.Imaging of abdominal hernias :Radiographics 1995;15:333-347
25. LeeGH, Cohen AJ. CT imaging of abdominal hernias. AJR Am J Roentgenol1993; 161: 1209–1213
26. Feldman M,Seharschmidt BF. Sleisenger and Fordtran's gastrointestinal and liver disease:pathophysiology/diagnosis/management 6th edition Philadelphia, Pa:saunders ,1998;324-328
27. Frank AJ, GoffnerLB,FruauffAA,LosardaRA.Caecalvolvulus:The CT Whirl sign.Abdominal imaging 1993;18(3):288-289.

28. FrencyPC,Stevenson GW, edsMargulis and Burhenne's alimentary tract radiology.5thedSt.Louis, Mo:Mosby-year book,1994;362-365,2059-2061.
29. Ogorek CP, Fisher RS: Differentiation between Crohn's disease and ulcerative colitis. Med Clin North Am 1994; 78:1249-1258.
30. Goldberg HI, Gore RM, Margulis AR, et al: Computed tomography in the evaluation of Crohn disease. AJR Am J Roentgenol 1983; 140:277-282.
31. Gore RM, Balthazar EJ, Ghahremani GG, et al: CT features of ulcerative colitis and Crohn's disease. AJR Am J Roentgenol 1996; 167:3-15.
32. Lee SS, Ha HK, Yang SK, et al: CT of prominent pericolic or perienteric vasculature in patients with Crohn's disease: Correlation with clinical disease activity and findings on barium studies. AJR Am J Roentgenol 2002; 179:1029-1036.
33. Shorter RG: Risks of intestinal cancer in Crohn's disease. Dis Colon Rectum 1983; 26:686-689
34. Gore RM, Laufer I: Ulcerative and granulomatous colitis. In: Gore RM, Levine MS, Laufer I, ed. Textbook of gastrointestinal radiology, Philadelphia: Saunders; 1994:1098-1141.
35. Levy AD, Hobbs CM. From the archives of the AFIP. Meckel diverticulum: radiologic features with pathologic correlation. RadioGraphics 2004; 24:565-587
36. Kusomoto H, Yoshida M, Takahashi I., et al. Complications and diagnosis of Meckel's diverticulum in 776 patients. Am J Surg 1992; 164:382-383

37. Konstantakos AK. Meckel's diverticulum–induced ileocolonic intussusception. Am J Surg 2004; 187:557-558
38. Bennett GL, Birnbaum BA, Balthazar EJ. CT of Meckel's diverticulitis in 11 patients. AJR 2004; 182:625-629
39. Lang EK, et al: Abdominal abscess drainage under radiologic guidance: Causes of failure. Radiology 1986; 159:329-336.
40. Ralls PW, et al: CT of inflammatory disease of the psoas muscle. AJR Am J Roentgenol 1980; 134:767-770.
41. Huang CJ, Pitt HA, Lipsett PA, et al: Pyogenic hepatic abscess. Ann Surg 1996; 223:600-609
42. Mathieu D, Vasile N, Fagniez P-L, et al: Dynamic CT features of hepatic abscesses. Radiology 1985; 154:749-752.
43. Radin DR, Ralls PW, Colletti PM, Halls JM. CT of amebic liver abscess.AJR Am J Roentgenol1988; 150:1297-1301.
44. Francis IR, Glazer GM, Amendola MA, Trenkner SW. Hepatic abscesses in the immunocompromised patient: role of CT in detection, diagnosis, management, and follow-up.GastrointestRadiol1986; 11:257-262.
45. Smith GS, Birnbaum BA, Jacobs JE: Hepatic infarction secondary to arterial insufficiency in native livers: CT findings in 10 patients. Radiology 1998; 208:223-229
46. Miller WJ, Federle MP, Straub WH, Davis PL.Budd-Chiari syndrome: imaging with pathologic correlation. Abdominal Imaging 1993; 18:329–335

47. Ito K, Mitchell DG, Honjo K, et al: MR imaging of acquired abnormalities of the spleen. AJR Am J Roentgenol 1997; 168:697-702.
48. Nelken N, Ignatius J, Skinner M, Christensen N: Changing clinical spectrum of splenic abscess. Am J Surg 1987; 154:27-33
49. Debnath D, Valerio D. Atraumatic rupture of the spleen in adults. J R CollSurgEdinb 2002;47:437–45
50. Fidler J, Paulson EK, Layfield L. CT evaluation of acute cholecystitis: findings and usefulness in diagnosis. AJR 1996;166:1085–1088 92.
51. Yamashita K, Jin MJ, Hirose Y, et al. CT findings of transient focal increased attenuation of the liver adjacent to the gallbladder in acute cholecystitis. AJR 1995;164:343–346
52. Grayson DE, Abbott RM, Levy AD, Sherman PM: Emphysematous infections of the abdomen and pelvis: A pictorial review. Radiographics 2002; 22:543-561
53. Baron RL, Tublin ME, Peterson MS: Imaging the spectrum of biliary tract disease. RadiolClin North Am 2002; 40:1325-1354.
54. Banks PA: A new classification system for acute pancreatitis. Am J Gastroenterol 1994; 89:151-152
55. Kalra MK, Maher MM, Sahani DV, et al: Current status of imaging in pancreatic diseases. J Comput Assist Tomogr 2002; 26:661-675.
56. Balthazar EJ: Acute pancreatitis: Assessment of severity with clinical and CT evaluation. Radiology 2002; 223:603-613
57. The revised Atlanta classification of acute pancreatitis: its importance for the radiologist and its effect on treatment. Radiology.262:751-764 2012

58. Kourtesis G, Wilson SE, Williams RA: The clinical significance of fluid collections in acute pancreatitis. Am Surg 1990; 56:796-799.
59. Yeo CJ, Bastidas JA, Lynch-Nyhan A, et al: The natural history of pancreatic pseudocysts documented by computed tomography. SurgGynecolObstet 1990; 170:411-417
60. Smith RC, Levine J, Rosenfeld AT: Helical CT of urinary tract stones: Epidemiology, origin, pathophysiology, diagnosis, and management. RadiolClin North Am 1999; 37:911-952.
61. Dalrymple NC, Casford B, Raiken DP, et al: Pearls and pitfalls in the diagnosis of ureterolithiasis with unenhanced helical CT. Radiographics 2000; 20:439-447.
62. Boridy IC, Nikolaidis P, Kawashima A, et al: Ureterolithiasis: Value of the tail sign in differentiating phleboliths from ureteral calculi at nonenhanced helical CT. Radiology 1999; 211:619-621.
63. Smith RC, Verga M, Dalrymple N, et al: Acute ureteral obstruction: Value of secondary signs of helical unenhanced CT. AJR Am J Roentgenol 1996; 167:1109-1113
64. Pickhardt PJ, Lonergan GJ, Davis CJ, et al: From the Archives of the AFIP: Infiltrative renal lesions: Radiologic-pathologic correlation. Radiographics 2000; 20:215-243
65. Gold RP, McClennan BL, Rottenberg RR: CT appearance of acute inflammatory disease of the renal interstitium. AJR Am J Roentgenol 1983; 141:343-349

66. Papanicolaou N, Pfister RC: Acute renal infections. RadiolClin North Am 1996; 34:965-995.

67. Wan YL, Lee TY, Bullard MJ, Tsai CC: Acute gas-producing bacterial renal infection: Correlation between imaging findings and clinical outcome. Radiology 1996; 198:433-438.

68. Yoder IC, Pfister RC, Lindfors KK, Newhouse JH: Pyonephrosis: Imaging and intervention. AJR Am J Roentgenol 1983; 141:735-740.

69. Kamel IR, Berkowitz JF: Assessment of the cortical rim sign in posttraumatic renal infarction. J Comput Assist Tomogr 1996; 20:803-806

70. Adam DJ, Bradbury AW, Stuart WP, et al. The value of computed tomography in the assessment of suspected ruptured aortic aneurysm. J VascSurg 1998;27:431–437

71. Arita T, Matsunaga N, Takano K, et al. Abdominal aortic aneurysm: rupture associated with the high attenuating crescent sign. Radiology 1997; 204:765–768

72. Mackiewicz Z, Molski S, Szpinda M, Jundzill W, Stankiewicz W. Retroperitoneal rupture of abdominal aortic aneurysms. J Mal Vasc 1998; 23:368–370

73. Williams DM , Lee DY , Hamilton BH , et al. The dissected aorta. III. Anatomy and radiologic diagnosis of branch-vessel compromise. Radiology 1997;203(1):37–44

74. Dähnert W Cardiovascular disorders: aortic dissection. Radiology review manual 5th ed. Philadelphia, Pa: Lippincott Williams & Wilkins 2003;607–609.

75. Williams DM , Joshi A , Dake MD , Deeb GM , Miller DC , Abrams GD Aortic cobwebs: an anatomic marker identifying the false lumen in aortic dissection—imaging and pathologic correlation. Radiology 1994;190(1):167–174.

S.NO	NAME	AGE	SEX	USG FINDINGS	CT. FINDINGS	ORGAN
						SPECIFIC
1	Kaliyamoorthy	67	Male	Acute appendicitis	Acute appendicitis	Bowel
2	Krishnamoorthy	65	Male	Appendicular mass	Appendicular mass	Bowel
3	Vellingiri	28	Male	Pancreas obscured by bowel gas ,mild ascities	Acute edematous pancreatitis	Pancreas
4	Sampathkumar	46	Male	Psuedocyst of pancreas	Cysto gastric fistula	Pancreas
5	Siva	32	Male	Mild left hun	Left lower ureteric calculus causing mild hun	Kub
6	Selvaraj	24	Male	Mild left hun	Left lower ureteric calculus causing mild hun	Kub
7	Dhanalakshmi	73	Female	Abdominal aorta aneurysm	abdominal aorta aneurysm with haematoma	Vascular(aorta)
8	Subbaiyan	70	Male	Abdominal aorta aneurysm	Abdominal aorta aneurysm with haematoma	Vascular(aorta)
9	Shanmugam	63	Male	?Abdominal aorta aneurysm	Abdominal aortic dissection	Vascular (aorta)
10	Saravanan	34	Male	B/l renal calculi, mild left hun	B/l renal calculi ,left ureteric stone ,left hun	Kub
11	Mayilsamy	58	Male	Left kidney abscess	Left emphysematous pyelonephritis	Kub
12	Meena	24	Female	Gas filled bowel loops	Sigmoid volvolus	Bowel

13	Sampath	53	Male	Dilated small bowel loops , midline structure obscured	Smvthrombous	Vascular (sma)
14	Ayyapan	38	Male	?Perforative peritonitis	Perforative peritonitis	Bowel
15	Subbaiyan	58	Male	Liver abscess	Liver and splenic abscess	Liver and spleen
16	Ponniselvi	28	Female	Dilated small bowel loops , midline structure obscured	Sigmoid volvolus	Bowel
17	Muthulakshmi	40	Male	Spleenic abscess	Splenic abscess	Spleen
18	Ajith	18	Male	Right paracolic abscess	Appendicitis with right paracolic abscess	Bowel
19	Praveenkumar	19	Male	? Left subphrenic collection	Left empyema with left subphrenic collection	Kub
20	Palani	27	Male	Left hun	Left vuj calculus causing left hun	Kub
21	Shajahan	37	Male	?Perforative peritonitis	Perforative peritonitis	Bowel
22	Kanniammal	70	Female	Left femoral hernia	Left femoral hernia	Bowel
23	Gurusamy	67	Male	Acalculuscholecystitis ,mild ascities	Diverticulosis of asc&des colon ,rent in asc.colon	Bowel
24	Nagarathinam	37	Male	Gas filled bowel loops	Sigmoid volvolus	Bowel
25	Pradeesh	35	Male	Gas filled bowel loops	Sigmoid volvolus	Bowel
26	Ponniselvi	28	Female	Fluid dilated small bowel loops	Closed loop bowel obstruction	Bowel
27	Ranjitha	20	Female	Grossly distended stomach	Sma syndrome	Vascular (sma)
28	Vadivel	36	Male	Mild ascities , dilated	Perforative peritonitis	Bowel

				small bowel loops		
29	Sekar	35	Male	Mild ascities ,midline structures obscured by bowel gas	Acute pancreatitis	Pancreas
30	Murugammal	40	Female	Left kidney enlarged, ?pyelonephritis	Left pyelonephritis with abscess	Kub
31	Manikkam	34	Male	Right hun	Right mid ureteric calculus causing hun	Kub
32	Vadivel	34	Male	Mild ascities , dilated small bowel loops	Acute pancreatitis	Pancreas
33	Padmanathan	45	Male	Dilated small bowel loops seen	Smathrobous	Vascular (sma)
34	Monanraj	39	Male	?Caecal abscess	Caecal abscess	Bowel
35	Balasubramanian	42	Male	Midline structures obscured by bowel gas	Aortic dissection	Vascular (aorta)
36	Rajalakshmi	60	Male	Dilated small bowel loops ,minimal bowel wall thickening	Bowel wall ischemia ,pneumobilia ,smathrombous	Pancreas
37	Varatharaj	31	Male	B/l mild pleural effusion,mildascities ,pancreas obscured	Acute edematous pancreatitis	Pancreas
38	Raman	60	Male	Acute appendicitis	Acute appendicitis	Bowel

39	Haridass	72	Male	Stomach dilated ,jejunojenunalintussception	Gastro jejunojejunalintusception	Bowel
40	Umavathi	42	Female	Acute appendicitis	Acute appendicitis	Bowel
41	Karuppasamy	28	Male	Rif probe tenderness	Acute appendicitis	Bowel
42	Menagadevi	53	Female	Left pyelonephritis	Left pyelonephritis with pararenal collection	Kub
43	Jawahar	44	Male	Small bowel obstruction	Jejunal obstruction	Bowel
44	Kunjappan	65	Male	Ileocolicintussception	Ileo colic intussception	Bowel
45	Malarvizhi	38	Female	Mild ascities ,pancreas obscured by bowel gas	Acute pancreatitis	Pancreas
46	Shanmugam	40	Male	Bowel wall edema seen ,minimal ascities	Smvthrombous	Vascular (smv)
47	Hariharan	18	Male	Acute appendicitis with intraperitineal abscess	Acute appendicitis with intra peritoneal abscess	Bowel
48	Kowsalya	15	Male	Right ileo psoas abscess	Right ileo psoas abscess	Retroperitoneum
49	Mumtaj	57	Female	Right kidney enlarged	Right pyelonephritis	Kub
50	Lakshmi	59	Female	Right kidney enlarged	Right pyelonephritis	Kub
51	Muthulakshmi	60	Female	Liver abscess	Liver abscess	Liver
52	Radhakrishnan	42	Male	Intestinal obstruction	Intestinal obstruction	Bowel
53	Patteswaran	38	Male	Small bowel mass ,?gist	Perforative peritonitis with gist	Bowel
54	Nagamani	60	Female	Mild bowel wall thickening	Bowel wall ischemia , pneumatosisintestinalis	Bowel

55	Radhakrishnan	33	Female	Acute appendicitis	Acute appendicitis	Bowel
56	Rangammal	67	Female	? Right emphysematous pyelonephritis	Right emphysematous pyelonephritis	Kub
57	Dinesh	22	Male	?Retro peritoneal abscess	Retro peritoneal abscess	Retroperitoneum
58	Murugan	32	Male	Intestinal obstruction with left inguinal hernia	Intestinal obstruction with left inguinal hernia	Bowel
59	Ramsudhakar	36	Male	Minimal bowel wall thickening in lif	Epiploicappendagitis	Bowel
60	Ayyanar	23	Male	?Sma thrombosis	Sma thrombosis	Vascular (sma)
61	Selvaraj	54	Male	Midline structures obscured by bowel gas	Renal artery thrombosis	Vascular (renal artery)
62	Govindharaj	70	Male	?Emphysematous cholecystitis	Emphysematous cholecystitis	Gall bladder
63	Perumal	67	Male	?Perforative peritonitis	Perforative peritonitis	Bowel
64	Appersamy	52	Male	?Perforative peritonitis	Perforative peritonitis	Bowel
65	Velmurugan	45	Male	Perforative peritonitis	Perforative peritonitis	Bowel
66	Selvi	42	Female	?Emphysematous pyelitis	Emphysematous pyelitis	Kub
67	Ramathal	55	Female	?Incisional hernia with sbo	Incisional hernia with sbo	Bowel
68	Saraswathy	45	Female	Sbo	Sbo with fallopian tube inflammatory mass	Bowel
69	Mohamed anifa	65	Male	Rectal sheath haematoma	Rectal sheath abdominal sheath hematoma	Abdominal wall

70	Rajamani	71	Male	Liver abscess	Liver abscess	Liver
71	Madappan	46	Male	Appendicular abscess	Appendicular abscess with smvthrombous	Bowel
72	Shanmugam	45	Male	?Perforative appendicitis	Perforative appendicitis	Bowel
73	Vadivel	46	Male	?Emphysematous right pyelonephritis	Emphysematous right pyelonephritis	Kub
74	Subbal	65	Female	Staghorn calculus	Staghorn renal calculus	Kub
75	Vellingiri	64	Male	Obstructed left inguinal hernia	Obstructed left inguinal hernia	Bowel
76	Thulasimani	55	Female	Bowel wall edema seen ,minimal ascities	Smvthrombous with bowel wall ischaemia	Bowel
77	Nagarajan	47	Male	Right perinephriccollection,lk not seen in left renal fossa	Right perinephricabscess,leftmalpositioned kidney	Kub
78	Siva	25	Male	Minimal ascities	Perforative peritonitis	Bowel
79	Sankar jones	25	Male	Dilated small bowel loops ,ascities ,pancreas obscured	Acute necrotizing pancreatitis	Pancreas
80	Venkateshwaran	42	Male	Minimal ascities ,pancreas obcured by bowel gas	Acute pancreatitis	Pancreas
81	Joseph	53	Male	Liver abscess	Liver abscess	Liver
82	Ramana	16	Male	Small bowel obstruction	Ileal stricture with small bowel	Bowel

					obstruction	
83	Parameshwaran	27	Male	Appendicular abscess	Appendicular abscess	Bowel
84	Palaniammal	63	Female	Rif probe tenderness	Acute appendicitis	Bowel
85	Sujan	28	Male	Rif probe tenderness	Acute appendicitis	Bowel
86	Nagamani	46	Male	Rif probe tenderness	Acute appendicitis	Bowel
87	Mylathal	57	Female	Rif probe tenderness	Acute appendicitis	Bowel
88	Nagarajan	55	Male	Pancreatic necrotic collection with ?Perispleenic collection	Pancreatic necrotic collection with peri splenic collection	Pancreas
89	Shakthivel	35	Male	Hepatic and spleenic abscess	Hepatic and splenic abscess	Liver and spleen
90	Perumal	52	Male	Acute appendicitis	Acute appendicitis	Bowel
91	Ravi shankar	46	Male	Staghorn calculus	Staghorn renal calculus	Kub
92	Ravi kumar	25	Male	Rif probe tenderness	Acute appendicitis	Bowel
93	Perarasu	50	Male	Perforative peritonitis	Perforative peritonitis	Bowel
94	Shah	33	Male	?Perforation	Perforative peritonitis	Bowel
95	Shalini	23	Male	Intussuception	Intussuception	Bowel
96	Beevi	34	Female	Apeendicular mass	Appendicular mass	Bowel
97	Ponnusamy	46	Male	Liver abscess	Liver abscess	Liver
98	Senthilkumar	25	Male	Acute on chronic pancreatitis	Acute on chronic pancreatitis	Pancreas
99	Sampathkumar	46	Male	Pancreatic necrotic	Pancreatic abscess	Pancreas

				collection		
100	Subramani	47	Male	Acute pancreatitis	Acute pancreatitis	Pancreas
101	Ramesh	36	Male	Acute pancreatitis	Acute pancreatitis	Pancreas
102	Mahesh kumar	48	Male	?Retro peritoneal abscess	Retro peritoneal abscess	Retroperitoneum
103	Parvathy	60	Female	Intussuception	Intussuception with polyps	Bowel
104	Jonhraj	28	Male	Acute appendicitis	Acute appendicitis	Bowel
105	Saraswathy	17	Female	Rif probe tenderness	Acute appendicitis	Bowel
106	Chandran	16	Male	Perforative peritonitis	Perforative peritonitis	Bowel
107	Ganesh moorthy	19	Male	Intestinal obstruction	Intestinal obstruction	Bowel
108	Prakasam	40	Male	Pyelonephritis	Pyelonephritis	Kub
109	Velathal	70	Female	Acute pancreatitis	Acute pancreatitis	Bowel
110	Maheshwari	27	Female	?Midgutvolvolous	Midgutvolvolous	Bowel
111	Baboon	18	Male	Small bowel obstruction	Small bowel obstruction	Liver
112	Vijay	28	Female	Liver abscess	Liver abscess	Liver
113	Vignesh	28	Male	Acute appendicitis	Acute appendicitis	Bowel
114	Indira	17	Female	Acute appendicitis	Acute appendicitis	Bowel
115	Leelakrishnan	47	Male	Liver abscess	Liver abscess	Liver
116	Shanthamani	32	Female	Torsion ovary	Torsion ovary	Ovary
117	Venkatachalam	33	Male	Intussuception	Intussuception	Bowel
118	Mohana	32	Female	Perforative peritonitis & peritoneal abscess	Perforative peritonitis & intra peritoneal abscess	Bowel

119	Nagaraj	48	Male	Liver abscess	Liver abscess	Liver
120	Nallammal	50	Female	Mesentricvolvolous	Mesentricvolvolous	Bowel
121	Chandran	48	Male	Spleenic abscess	Spontaneous splenic rupture	Spleen
122	Kanniammal	70	Female	Right obstructed femoral hernia	Right obstructed femoral hernia	Bowel
123	Vellingiri	57	Male	Retro peritoneal abscess	Rt.emphysematouspyelonephritis &retro peritoneal abscessrad.sugg	Kub
124	Chellappan	72	Male	Appendicular abscess	Appendicular abscess	Bowel
125	Palaniappan	50	Male	Gas filled bowel loops	Sigmoid volvolus	Bowel
126	Vellingiri	64	Male	Obstructed right inguinal hernia	Obstructed left inguinal hernia	Bowel